SEPTEMBER, 1950

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CONVENTION NUMBER

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Pages I-XX

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Fifteenth Annual Convention
HOTEL STATLER
New York, N. Y., 9, 10, 11 October 1950

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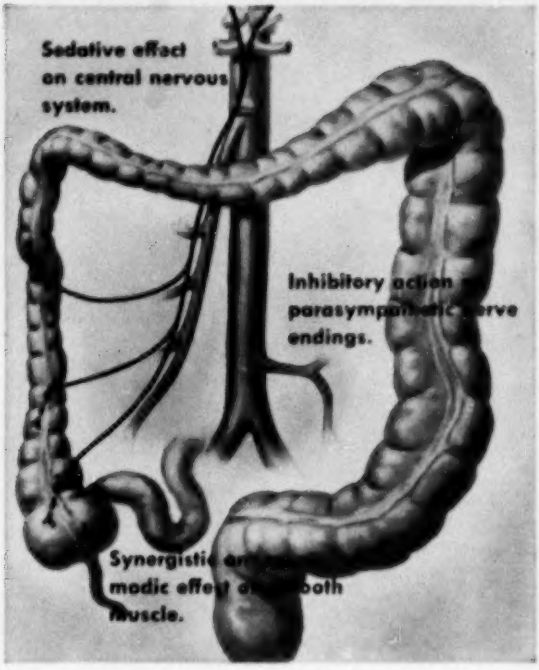

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(Incorporating the American Journal of Gastroenterology)

The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada

Volume 17 SEPTEMBER, 1950 Number 9

CONVENTION NUMBER

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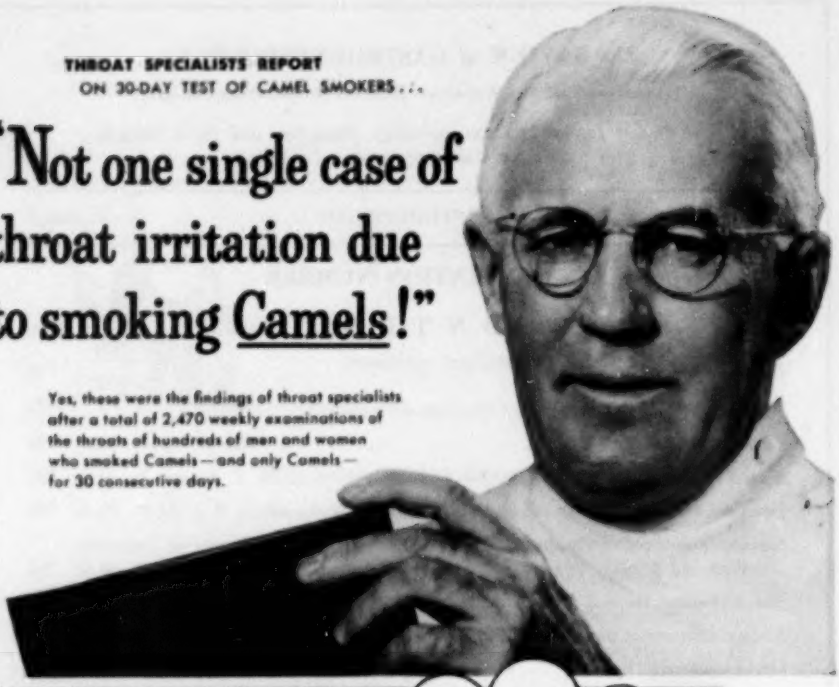

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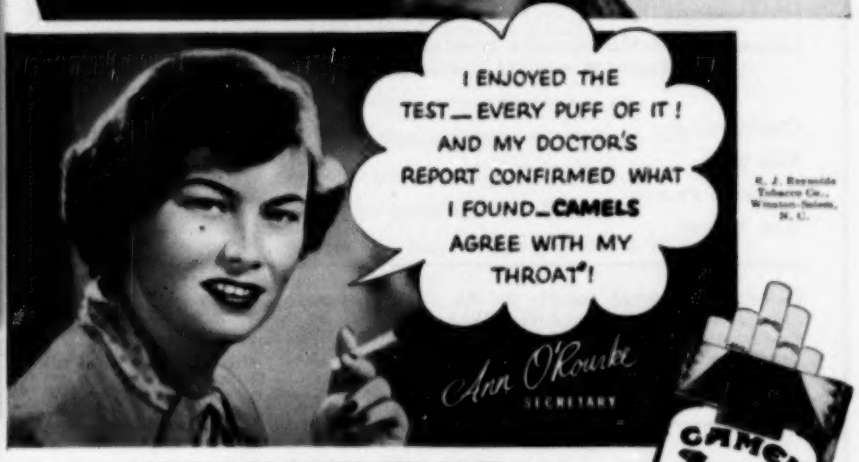
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1. Weiss, S., et al.: Rev. Gastroenterology 16:501-509 (June) 1949.

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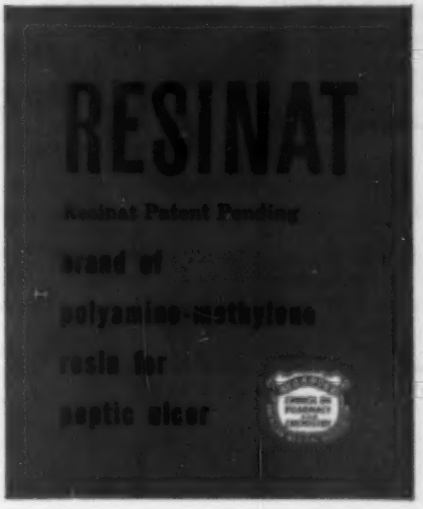

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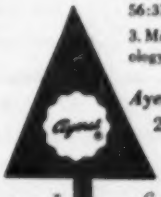
1. Kraemer, M.: Postgrad. Med. 2:431 (Dec.) 1947.
2. Kraemer, M., and Siegel, L. H.: Arch. Surg. 56:318 (Mar.) 1948.
3. Martin, G. J., and Wilkinson, J.: Gastroenterology 6:315 (Apr.) 1946.



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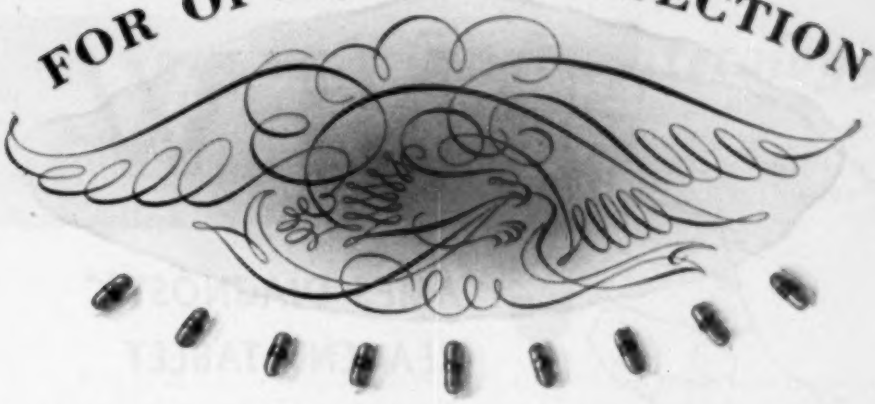

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Volume 17 SEPTEMBER, 1950 Number 9

ULCERATIVE COLITIS*

FRANK H. LAHEY, M.D.†

Boston, Mass.

First of all, I do not believe that this subject is so controversial from the point of view from which I wish to approach it. There is, I believe, no question in our mind that a majority of the patients with ulcerative colitis are manageable medically. There has arisen a side to this question, however, which promotes some doubt about the wisdom of medical management; I would not insist on the point of view that might be deduced from the high incidence of malignancy that all cases of ulcerative colitis ought to be treated surgically. On the other hand, I will try to present to you in the course of this discussion at least some data which are now becoming more and more available concerning the increasing incidence of malignancy in patients with ulcerative colitis who retain their colons, particularly when the disease has reached the polypoid stage.

I think that it might be helpful to present to you perhaps a little biased point of view regarding some of the emergencies which occur in connection with ulcerative colitis. You who practice gastroenterology, by natural circumstances see the cases which are not the most urgent type, because the patients in very acute episodes of ulcerative colitis, from the point of view of toxicity, that is, with high temperatures, nausea, vomiting, multiple stools, with blood and pus, and with distention, very often are sent by their family physicians directly to the surgeon. Likewise, the patients with massive hemorrhage often go directly to the surgeon, or into a group such as ours or other groups everywhere all over the country, who are interested surgically in ulcerative colitis. There is still another group of patients of this sort, that is those patients with obstruction and perforation, who go directly to the surgeon. Still another group of patients who do not go to the surgeon early enough is composed of those who have joint infections.

Those are the exaggerated cases of ulcerative colitis that we see. On the other hand, you could rightly say: "You do not see in large numbers the type of patients whom we see, who do quite well medically." We are now seeing them. We have had between seven and eight hundred patients with ulcerative colitis in our own

*Read before the Fourteenth Annual Convention of the National Gastroenterological Association, Boston, Mass., 24, 25, 26 October 1949.

†Director, Lahey Clinic; Surgeon, New England Deaconess and New England Baptist Hospitals, Boston, Mass.

management, and most of them are doing quite well medically. It is from this large group that we have been able to draw both experiences, that is, the experiences with the nonsurgical management, and the experiences dealing with the surgical emergencies that occur if one deals with a large number of patients with ulcerative colitis.

I can perhaps accomplish most if I can promote in your mind the things that are active in mine, and they are that certain things are true about this disease.

First, emergencies do arise that cannot be handled medically.

I know there have been statements to the effect that surgeons have been over-enthusiastic. I have no doubt in certain places that has been true. There have been statements to the effect that patients have probably been made very unhappy from surgical procedures. That was true particularly with ileostomy, but should not be now. There have been statements to the effect that hemorrhage is never an urgent situation in ulcerative colitis. That is not true, because we see it often and it is an urgent complication that must be dealt with aggressively; otherwise fatalities occur.

Many of the disagreements are based upon something that I said at Atlantic City at two separate meetings. I discussed a paper at one of the sections on hematology in relation to blood dyscrasias and splenectomy, and there I said something that is also so true of ulcerative colitis and ileostomy. It is: the mortality of ileostomy or the mortality of splenectomy in patients with blood dyscrasias is not in the hands of the surgeon; it is in the hands of the hematologist, and in the hands of the medical man and gastroenterologist because we now know how to do ileostomies very well. We can do ileostomies that will function, that will not prolapse, in which the skin will not ulcerate around the ileostomies, and which will be well controlled. We have measures to control them and we have ileostomy bags. To illustrate, I brought with me a patient upon whom we have done a total colectomy and abdominosacral resection, who is wearing a Rutzen bag, in order that I can take her up on the stage and demonstrate that she can wear any kind of tight-fitting clothes, even including a bathing suit, and in no way be disfigured by an ileostomy bag.

She can tell you that the bag will not leak. She can bathe, swim, play golf, do anything in the world she wants to do; in other words, we wanted to demonstrate to you that ileostomy should no longer be something that frightens you or the patient, so that you wait until the patient is in extremis.

As we have repeatedly stated, we have had a very fortunate experience in being able to do ileostomies early in the history of the surgical management of ulcerative colitis. We now have two groups of cases, an early group and a later group, in which we can compare mortality. We performed ileostomies on a consecutive early group of 145 cases with a mortality of 22.3 per cent. That is a prohibitive mortality, but we have now done 70 ileostomies with a mortality under 4 per cent.

That is not due to the fact that we do ileostomies better and manage them better than we used to, but it is due to the fact that the cases can be done earlier;

that they are not done when the patients are in extremis. That is not entirely because we have educated the New England public or the groups who have been interested in this subject, to send them in. It is because we have something to offer these patients with an ileostomy. We can promise them beyond any measure of doubt that they can marry, they can have babies, that they can do anything they want to after their total colectomies, abdominosacral removals of their rectums and ileostomies.

People with ileostomies in a way form clubs; they all know each other. This patient here with me knows almost everybody who has had an ileostomy around her time. They meet each other if they come to the clinic and they visit. They know, as well as many of us in the clinic who have been interested in this do, what the complications with ileostomy are, how well they can do with them, and what the limitations are.

You know the indications for ileostomy (Table I) as well as I do. The only thing I should like to urge is: do not wait until an acute fulminating case reaches the stage where no surgeon, no matter how much experience he has had with ulcerative colitis, can do an ileostomy without a high mortality.

TABLE I

INDICATIONS FOR ILEOSTOMY

1. Acute fulminating cases
2. Medical failures
3. Massive hemorrhage
4. Subacute perforations, abscess, peritonitis or fistulas
5. Obstruction
6. Polyposis
7. Malignant degeneration
8. Joint infection
9. Incontinence from rigid anus

We have repeatedly said if you wait until these patients reach the acute fulminating stage in which the gastroenterologist, the family doctor, the patient, and the patient's family all realize that if an ileostomy is not done the patient will die, that ileostomy cannot be done without a mortality of 25 per cent. That is because if we open that patient's abdomen and touch the colon, or do almost anything within the peritoneal cavity, the patient will get peritonitis by permeation, because the colon is literally permeated with organisms and is edematous.

We have just had a case which Dr. Cattell was doing in the next room to mine, in which the intestine was of the consistency of wet blotting paper. If they are allowed to go on to these stages, then, of course, nothing can be done.

We do not want these patients to endure too many medical failures before coming to surgery, particularly when you look at this patient here with me, and the dozens of others like her who can be so completely rehabilitated. We must not go on with medical failures until some of these complications result that are irreversible. One complication which is not recognized often and early enough is joint changes.

These people who have had constantly infected joints because of retained and infected colons cannot be restored to health. We cannot restore their joint

motion after they have had cartilage erosion. For that reason we would like to urge that it is very unlikely that any of these patients with ulcerative colitis who have joint involvement can be managed medically. It is not a question of hemorrhage, of obstruction, of toxicity, or of perforation; it is a question of chronic repeated infection of joints. This complication should be an outstanding indication for ileostomy, total colectomy, and abdominosacral removal of the rectum if there is extensive involvement of the large bowel.

Massive hemorrhage has been a very serious problem. I wrote an article on this subject in the July 1949 issue of the Bulletin[1]. We used to say that we would never dare to do a total colectomy and abdominosacral resection in one stage, particularly in a patient who had had ulcerative colitis for several months and had been so intoxicated and depleted by it that she had lost weight and was a poor risk. We have recently, within the last year, had to do an abdominosacral and total colectomy in one stage on a young girl who was literally bleeding to death, in whom we had tried everything, even filling the colon with plasma. She had had multiple transfusions. The ulcerative colitis was uncontrollable and it was a choice of total colectomy and an abdominosacral resection or a fatality. Fortunately she recovered. We have had that to face a number of times.

We would like to urge upon you not to let these patients who do have massive hemorrhage with ulcerative colitis get into such stages that when you decide to do colectomy, they have a very limited chance of surviving because of their depleted condition. If they have repeated hemorrhages you must seriously consider the control of the bleeding by means of either partial or complete colectomy.

As those patients go on to subacute perforations, abscesses, peritonitis, or fistulas, the question is raised, what about the patients who have multiple fistulas, and is it wise to continue with medical management?

Sometimes these patients have multiple fistulas in great variety. They are found in other sections of the bowel. They perforate in the small intestine and, of course, many patients have a so-called pepperpot anus—multiple fistulas about the anus. By the removal of such colons and rectums, the institution of ileostomy, and the use of a Rutzen or Torbot bag, you can abolish these fistulas completely as they then all heal.

What can you do with the patients who have subacute perforations and multiple abscesses? Colectomies can be done on many of them because they have such tremendous local resistance. I recall some of our experiences with colectomies done for ulcerative colitis with perforation and abscesses, and what these patients have survived—it is amazing.

We can recall a young girl who came to us before we knew how to handle ulcerative colitis, upon whom we had done an ileostomy. This was in the days before we had the Rutzen bag. She discharged everything upon her abdominal wall. It digested away her ileostomy, the ileostomy loosened from the abdominal wall and retracted into the free peritoneal cavity. She rode from Taunton to Boston with the ileostomy discharging into the peritoneal cavity. We washed out the cavity and she recovered. These patients have tremendous local resistance to these

local infections, and you can do things to them such as resections that you could never do upon unvaccinated patients.

Obstruction within the retained colon may occasionally result if the patient has an ileostomy and a retained colon. I believe it will soon be universally accepted that once an ileostomy is done, that patient will be better off with a colectomy and an abdominosacral removal of the rectum. If the colon is a true lead-pipe colon, there are repeated areas of cicatrization and obstruction. As a result of these multiple cicatrized areas, greater in one place than another, pus pockets will develop from which so often will arise these infections of the joints that produce destruction which is irreversible and irreparable. These are the cases for which I recommend earlier ileostomies and colectomies. Many of the obstructions come after the ileostomy is done and the colons are put at rest. In 80 per cent of all patients who have ulcerative colitis the rectum is involved; therefore, it is very unlikely that you will ever be able to restore the fecal stream in many of these

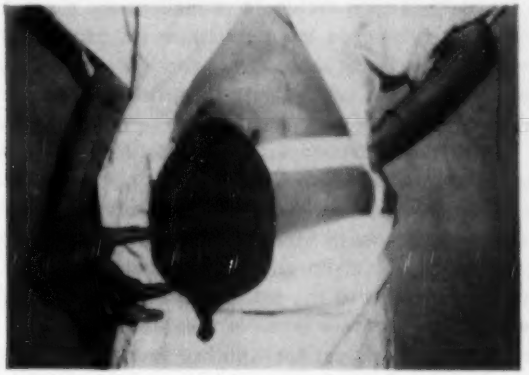

Fig. 1—The ileostomy bag in place. Note the spigot at the bottom of the bag with which to empty it and also note the flatness of the bag.

cases. It is for that and other reasons, that I would urge that if your patients have ileostomies, their colons also should be removed.

If an ileostomy is performed and the colon taken out, the character of the ileal discharge will immediately change. It will no longer be entirely liquid most of the time. In the majority of cases it will thicken and become semi-solid and much easier to manage. Probably this is because the terminal ileum, which is left behind, takes up some of the fluid-absorbing function which belongs to the right colon when it is normal. Likewise it is true that if these colons are removed there will not, as already stated, be the secondary infections of joints which occasionally occur in these colons even after ileostomies. Most important, you will not have malignancy which is being found with increasing frequency in the retained colon.

In our experience cancer of the colon will develop in 5 per cent of all patients who have ulcerative colitis. In very few of those patients who develop cancer will the diagnosis be made early enough so that an operation can be done which will give a good five-year survival rate. We have had 18 patients with ulcerative colitis

in whom cancer of the colon or rectum developed, and only 2 are alive. They are alive only three years after operation. In both of these cases cancer was found by the pathologist incidentally on microscopic examination when he was examining the colon.

Unfortunately; the urgency of the symptoms of ulcerative colitis are such that they will obscure the superimposed symptoms of a carcinoma. A diagnosis of carcinoma in a patient with ulcerative colitis can be made only under these conditions: when a mass can be felt, when there is an unexplained hemorrhage of bright blood and when there is an obstruction. By the time these conditions are present the lesion will be so large that it will not be curable, particularly in an inflamed colon.

In addition to that we know, from our own experience, that the longer patients with ulcerative colitis retain their colons, the higher is the incidence of the development of malignancy. It is probably true that in the patients who have had an ulcerated colon for ten years, the incidence of malignancy may rise to 30 per cent.

I do not believe it is true, as has been suspected, that the incidence of carcinoma in ulcerative colitis is the same as the incidence of polyp, which probably is 5, 6 or 7 per cent, and that carcinoma results from malignant degeneration of the polyp. I believe it is due to a different type of process. It is due, we believe, to the frequent hyperplasia and metaplasia that goes on in these colons, with their repeated changes of acute episodes and resolution.

A majority of these patients with ulcerative colitis can be treated medically. Of 327 cases that were followed from two to fifteen years, it was found that medical management brought about either marked improvement or complete relief of symptoms in two-thirds of the patients with the milder forms and in one-third of those with the more severe forms of the disease.

Finally, before I show this patient who has been good enough to come here, I should like particularly to speak about how these operations are done. These patients used to have to go through multiple stages. We used to do an ileostomy and then wait three months. We then resected the right colon and transverse colon to the splenic flexure, and waited three months. Then the splenic flexure, descending colon and the sigmoid were removed, and we waited three months. That rarely happens now. With greater experience we now do the ileostomy and wait until it is well established. We then take the colon out down to the sigmoid and wait three months, and then do an abdominosacral resection.

We used to fear greatly that these patients would decide to have a baby. We advised them strongly against it, and they, much against our advice and judgment, have had babies and proven to us that we were wrong. They have had babies between the various stages of the operation—between the first and the second stages, between the second and third stages, and after the final stage, and so far nothing has happened to any of them.

Before I show the patient, I will say that she will be glad to answer any questions you want to ask. She has dedicated herself, in a measure, to patients with ileostomies, and she is willing to come in here because she thinks that if she

can show you that these patients can be completely rehabilitated, it will be a contribution from her in repayment for what she feels has been done for her.

To summarize these remarks:

(1) Ileostomy can now be done with complete satisfaction.

(2) The patients can live with complete comfort and participate in every activity.

(3) Do not delay resection of the colon until the patients get joint changes.

(4) Do not let these patients get in desperate condition from intoxication before the ileostomies are done.

(5) Do not let the patients with hemorrhage continue to bleed too much before the colectomies are done. Colectomy promptly removes the danger of hemorrhage and rehabilitates them.

(6) Finally, keep in mind that there is growing evidence that the patient with ulcerative colitis who retains his colon is a candidate for an increasing percentage of carcinomas.

Now we will present the patient. Here is the Rutzen bag (Fig. 1). She wore a tight-fitting dress purposely, and any of you can see the bag if you wish.

The bag is perfectly flat, has a spigot in the bottom, and can be closed with an elastic band. The cement used is mostly Latex with a little zinc oxide in it, and it is put on just the way you patch a tire. It is put on the bag and on the skin and, when it is tacky, the bag is placed on the skin and it is tight. It has a couple of little clips on the side to which a belt is attached. The patient can loosen the end and go to the toilet and dump the contents, and she changes it once in twenty-four hours. She keeps an extra bag. She will let you see it and you can examine the bag.

I feel a little like a patent medicine man showing this patient, but I do want to give you confidence from actual demonstration that these patients can have ileostomies and live, I believe, more happily even than the patients with colostomies and they do very well. They have no skin irritation. You read in the literature that these ileostomies need to be skin-grafted. That is not necessary at all. All you need to do is measure the ileostomy opening and these bags are so made that they fit accurately with no skin exposed.

REFERENCE

1. Lahey, F. H.: Massive Hemorrhage in Ulcerative Colitis. Lahey Clin. Bull. **6**:130-131, (July), 1949.

Discussion

Dr. William E. Browne (Boston, Mass.):—If anyone here would really like the job of discussing Dr. Lahey's paper I would be perfectly pleased to sit down. I believe from what I am told if you applied now for a ticket for South Pacific currently playing in New York, you might get one within six months' time. I am told, too, one of the fine selections in that play is "Some Enchanted Evening". I do not know the words but I do know I invariably find myself enchanted, so to speak, in listening to the words of Dr. Lahey. I have heard him discuss this subject before and indeed he has made it just as much, if not more, interesting this

morning. You know there isn't one single sentence which he has uttered with which one could be in any real disagreement. In substance he has said: "The vast majority of patients afflicted with colitis of one form or another are well handled by medical management". He has pointed out three or four things which are important to bear in mind in connection with those cases of colitis—ulcerative colitis —which are really surgical problems. I am sure he has made it very clear what surgery can do for many of these patients.

I would judge from the large number here present this morning that Dr. Morrison must in a way feel somewhat repaid for his really untiring efforts in making this national meeting a success. He has told me of the distinguished Englishman who will be one of the speakers at tonight's dinner. A number of years back when Dr. Fred B. Lund was President of the American College of Surgeons, at the time when the late Sir Arbuthnot Lane was advocating removal of the large bowel for obstipation the then Sir Berkeley Moynihan—later Lord Moynihan—was here in Boston and spoke at meetings of the American College of Surgeons. He was the house guest, while here, of one of the great surgeons of Massachusetts, the late Samuel Mixter. He had a very fine herd of Guernsey cows. Dr. Mixter and Sir Berkeley were out for a walk that afternoon and covered pretty well the terrain of Dr. Mixter's farm. At the meeting that night somewhat facetiously Sir Berkeley referring to Sir Arbuthnot Lane, and recognizing the existence of many critics of Sir Arbuthnot's procedure, did say he wasn't too sure as to the number of humans in the United States who were troubled with intestinal constipation or obstipation, but after having been out with his friend Mixter this afternoon he was quite sure that cows were in no way afflicted.

I have referred to three Englishmen thus far. There was another, the late John Henry Newman. In his idea of a university, writing on what education did for one, he said in substance that it enabled one to form opinions and judgments and to express them—"it enables one to have a clear conscious view of his own opinions and judgments, a truth in developing them, a force in urging them and an eloquence in expressing them". Dr. Lahey has shown these attributes in his remarks on ulcerative colitis. Among other things Newman, in regard to education, said, "education enables one to know when to speak and when to be silent. It enables him to ask a question pertinently and gain a lesson seasonably when he has nothing to impart himself". I have nothing to add to Dr. Lahey's remarks. It is quite obvious that he has intentionally not considered many things which are of great importance in making a correct diagnosis in various forms of colitis. He has forcefully presented for consideration facts which must be kept in mind in the handling of these very difficult problems.

I would like to ask him just one question, and it is this. When you have decided a patient with colitis is a surgical problem; when you have first done an ileostomy and later, depending upon what further studies including x-ray reveal, you have removed part of the large bowel, or all of it, down to the lower sigmoid; do you have any rule which you invariably follow as regards later on closing the

ileostomy and establishing continuity between the terminal ileum and the upper segment of the rectum? I have profited by Dr. Lahey's remarks. Again I would have you know of Dr. Morrison's untiring efforts in making this convention a success.

Dr. Saul Schapiro (Brooklyn, N. Y.):—To listen to a paper by Dr. Lahey is always a distinct privilege, but to discuss a paper of his is a rare honor. This society is indeed fortunate to have the opportunity of hearing him and will benefit from his vast experience.

There are few diseases that tax the patience and ingenuity of the doctor more than does ulcerative colitis. However, the problem is becoming somewhat easier with the establishment of medico-surgical teamwork. Chronic ulcerative colitis represents a multiplicity of diseases and we must remember this when discussing the matter so that we all talk about the same treatment for the same type of ulcerative colitis; that is, is it acute, subacute, or chronic? Is it infectious, non-infectious, psychogenic, a nutritional deficiency, an allergy, etc.? These types of colitis, each having a different pattern, require different modes of treatment, and that is when the medical and surgical teamwork becomes important.

With constant exchange of ideas and experience between medical and surgical workers, there has resulted at least as a working basis, a consolidation of thought and general understanding as to what constitutes the medical disease, when it becomes a surgical disease, and what type of surgery might be best performed.

Because of the advances of pre- and postoperative care, more patients with ulcerative colitis are being treated by radical surgical procedures. It is now realized that even after ileostomy, the colon, with its extensive changes never reverts to normal, and reestablishment of continuity of the bowel can seldom be restored without danger of recurrence. Further, the diseased colon acts as a focus of infection, preventing complete restoration of health. Also the possibility of polyposis and malignant degeneration always exists.

As surgery has become safer, there has been an increasing tendency to follow ileostomy with resection of the colon. The results of colectomy have been relatively good. It remains to decide which is best, (1) permanent ileostomy with colectomy including abdominal perineal resection; (2) colectomy with ileorectostomy or ileoanostomy; or (3) permanent ileostomy with right ileocolectomy in the first stage, followed later with resection of the remaining colon.

Regarding the latter, a few days ago I had a personal conversation with Dr. Charles Ripstein, coworker with Dr. Miller. To date they have had 54 cases, in 35 per cent of which they carried the colon resection down to the sigmoid while doing the ileostomy; that is, in the first stage the distal sigmoid loop was brought out to the abdomen, and the remaining sigmoid and rectum were dealt with later. Of the 54 cases done at this one stage down to the sigmoid, two died, one of perforation, and the other of massive hemorrhage, both occurring in the remaining colon.

In seven cases under my observation with ileorectostomies, three had fairly good results, but three had such severe symptoms from the retained rectum, they begged to have the ileostomy restored.

I can offer no other contribution to the valuable and comprehensive information given us today by Dr. Lahey except to emphasize the importance of medico-surgical teamwork. Though no definite criteria can be given by which one can decide when medical treatment has failed and surgery should be done, there are certain opinions on which there seems to be general agreement:

1. That ulcerative colitis remains primarily a medical problem. Most patients have a very mild type of colitis in which the symptoms are not severe and the disease constitutes an inconvenience rather than a disability. They rarely develop complications, do well medically, and do not require surgery unless there are clearcut complications or progressive intractability.

2. That ulcerative colitis is a medical disease in 75 per cent of the cases, and surgical in approximately 25 per cent.

3. That since surgery has become safer, more cases come to operation at the properly chosen time for the carefully selected cases.

4. That the acute, fulminating type is a medical disease in which surgery is disastrous and should be avoided unless it is done in the early stages, and even then the patient must be adequately prepared with the best supportive measures. In this type the recently described medical ileostomy may have a place.

5. That the decision to do ileostomies, especially in the acute fulminating type, take some of the following factors into consideration: rising temperature, vomiting, low serum protein, poor appetite, loss of weight and strength, uncontrollable bloody diarrhea and anemia despite repeated blood transfusions, massive hemorrhages, etc.

6. That ileostomy rehabilitates the chronic invalid and saves lives.

7. That an ileostomy should not be closed.

8. That ileostomy be performed with intent to follow with colectomy but not before at least four months have elapsed.

9. That the indications for colectomy include frequent attacks of bleeding and diarrhea associated with joint and skin changes, polyposis, obstructions, severe perianal or perirectal infections, impending carcinoma, incontinent anus.

10. That although many patients with segmental colitis become symptom-free on a medical regime, if the clinical and x-ray findings show progression of the disease, resection is advisable before extension of the colitis precludes anastomosis.

11. That colectomy should be as complete as possible.

12. That until recently the colectomy terminated with ileosigmoidostomy or ileorectostomy but now the trend is to remove the diseased rectum also, ending either with a permanent abdominal ileostomy or ileoanostomy with or without sphincters. It remains to be determined whether the ileoanal anastomosis gives the patient a proper functional result.

Dr. Edward T. Whitney (Boston, Mass.):—I should like to add to what both Dr. Browne and Dr. Schapiro have said about Dr. Lahey's paper.

Dr. Lahey's experience with the management of something over 700 cases of ulcerative colitis is a major feat in itself, as the number of cases coming under the care of any one physician or group of physicians is usually limited to a small

number in any one year. This may be one of the reasons why so little is known concerning the disease and why so many cases do require, at some time or other, an ileostomy.

Life after this procedure is not to be anticipated with pleasure, though perhaps this lady and Dr. Lahey would say otherwise, but, on the other hand, neither is life markedly pleasurable with the so-called chronic form of this malady. The fact that we cannot routinely throw a patient into remission and keep him there is an admission on our part that we still know very little concerning the etiology, for usually when the etiology of a disease is known, modern medicine can find some sort of cure or preventive.

As Dr. Lahey's paper indicates, investigation is being made, and progress is steadily being had, albeit most of such investigation and progress has to be of the clinical or therapeutic variety by way of the trial and error method. Such investigation has substantially followed three seemingly isolated pathways with surgery becoming necessary in case of failure. Because of the partial success obtained by each one of the three lines of endeavor, they all require serious examination and consideration.

Firstly, the psychiatric approach has had some such surprisingly excellent results that a firm foundation has been laid for considering the condition as of psychosomatic origin in many cases, especially when it can be pointed out that emotional pressure is frequently found to be the activating factor in these cases, and that psychotherapy produces a remission.

From a physiological standpoint it would appear that in such patients chronic emotional tension overactivates the parasympathetic side of the autonomic nervous system to such an extent that hypermobility and hypersecretion ensue, and when we say "parasympathetic system", we mean more than just the parasympathetic nerves; we mean brain, cholinergic nerves of both parasympathetic and/or sympathetic origin, acetylcholine, cholinesterase, histamine, certain enzymes, certain hormones, and even certain hematopoietic agents or processes.

The second line of investigation was instituted by one of our colleagues, Dr. Robert Ehrlich, a member of this Association, who, in the American Journal of Digestive Diseases, for September, 1947, entertained the assumption that in this disease there was a loss of an intrinsic factor which normally prevented autodigestion of the colonic mucous membrane. He administered desiccated hog's stomach (that being the only known and available source of such a factor at that time), and except in those cases found to be allergic to pork, he could produce remissions at will.

His work was shortly confirmed by Haskell and Friedman, who reported in the December, 1948, issue of the American Journal of Surgery, a series of cases treated with an extract obtained from duodenal substance.

Better, more potent, and cheaper sources of supply of this factor are being discovered, which may answer some criticism of this form of therapy. The question promptly arises: Is it possible that the overstimulation of secretion induced

by a hyperactive parasympathetic nervous system so exhausts the supply of this intrinsic factor that autodigestion ensues?

The third line of investigation is the old one, namely, that of infection of the bowel wall so long advocated by Bargen and still being reported on or against in the literature, and as mentioned by Dr. Lahey today. Certainly we know that the use of the nonabsorbable sulfa drugs is a needed and useful measure in producing some remissions. This poses a second question, namely: Is it possible that such an infection becomes superimposed on an autodigestion brought about by exhaustion of an intrinsic factor during chronic hyperparasympathetic activity?

For a long time now it has seemed to us that until this latter question has been refuted, adequate management of this disease necessitates the institution of *all* of these three forms of treatment early in its course; that is, before the glands producing the intrinsic factor have become so exhausted by overstimulation that atrophy has taken place, thereby subjecting the patient either to the constant need for an artificial supply of this factor, or for the surgical removal of his colon.

Dr. Lahey and his coworkers are to be commended for their farsighted approach to this problem as revealed in his paper today.

Dr. Norman Strauss (New York, N. Y.):—I should like to ask Dr. Lahey if the introduction of streptomycin or chloromycetin have in any way influenced the course of the disease either pre- or postoperatively?

Dr. Emil Granet (New York, N. Y.):—I should like to ask Dr. Lahey as to the experience at the clinic with perianal fistulas, specifically the approximate percentage, and the management of those cases with the colon retained after ileostomy. The perianal infections in our cases have been terrible in extent and more common that the reported statistics.

Dr. Horace W. Soper (St. Louis, Mo.):—I wish to say that I completely agree with Dr. Lahey in his ileostomies instead of colostomies. We have had in our experience very severe complications following colostomies in ulcerative colitis because later on complications occur in which colectomy must be done after the colostomy has been there for some time. Then when the surgeon gets down there, you find the ureter imbedded in that colon, and in the surgeon's attempt to free it, of course, the patient dies later of peritonitis.

I want to say one thing about the early diagnosis of ulcerative colitis. It usually begins low down in the colon. In the large majority of cases it can be diagnosed by means of the sigmoidoscope. You look up there and you find the characteristic lesions of ulcerative colitis.

Dr. Lahey:—Tell them what they are like.

Dr. Soper:—They are ulcerative in character. They are disseminated, not always completely involving the colon, but they are essentially ulcerative. They are absolutely different from the ordinary type of catarrhal colitis, absolutely different, so you can make an early diagnosis.

For twenty years I have used a treatment for early ulcerative colitis which has been very effective, and that is the simple mineral oil retention enema, six to nine ounces being given at night, and allowed to remain in the colon all night.

Why? It is not a culture medium. No bacteria will grow in the presence of mineral oil; after six weeks or so of this course of treatment, your colitis is cured.

It is in our late cases, of course, that we must resort to ileostomies and resections following resections, as Dr. Lahey has said.

Dr. Frank H. Lahey (Boston, Mass.):—In answer to Dr. Strauss, the introduction of streptomycin and chloromycetin has not in any way influenced the course of the disease.

In answer to Dr. Granet, our experience with perianal fistulas in cases in which the colon has been retained has been terrible, too. I do not think it is feasible to control fistulas that originate from a retained infection.

I should like to say one thing about the fistulas and that is, you can remove these rectums in the presence of severe infections around the anus. There is almost nothing that can happen to them that does not make their removal possible and, strangely, the posterior wounds heal quite well.

You must not expect the posterior wounds not to cause you trouble later on. They often break down repeatedly for several months in small areas but they ordinarily heal nicely. We expect our patients who have had abdominosacral resections for cancer of the rectum or ulcerative colitis to leave the hospital in two weeks. We close them all posteriorly except for a small drain. No longer are they packed and left open. It is a saving in time and suffering and also in mortality.

Before I answer the other questions, I should like to bring one thing to you that I am quite sure will be helpful to you as gastroenterologists, and that is trial ileostomies. We have done 25 trial ileostomies. In early cases in which the patients do not do well medically we can now do an ileostomy and later restore fecal continuity. We will not restore the fecal stream until the following conditions are met. First, the disease must completely disappear; that is, there must be no sign of the disease such as diarrhea, fever, blood and pus. Second, haustrations in the colon must return. There is no logic in restoring colons that do not have haustrations. Third, the rectal mucosa, as Dr. Soper has just said, must become normal; it must be nongranular, it must not bleed and it must be flexible. Unless the mucosa is normal, restoring the fecal stream will reawaken the disease. Fourth, you must tell the patient that in spite of fulfilling all of these three requirements completely, the disease will be reawakened in 33 per cent of the cases when the fecal stream is restored, and these patients will have to have another ileostomy. But when you realize that two-thirds of the patients have received benefit from the sidetracking and then have had their fecal stream restored, it is worth keeping in mind.

The question asked was whether we ever leave segments of the bowel and do anastomosis and restoration of the fecal stream. Yes, we do when the disease is segmental, but I should also like to say that it is impossible for me to tell from the outside of the colon how far the process extends on the inside. Therefore, if there is segmental involvement, you must not do a limited resection. You must resect the bowel 2 and even 3 feet ahead of what appears to be the end of the process.

As to the acute fulminating cases, Dr. Schapiro, I am quite sure we would agree except that I do not want you to get the impression that the patients with the acute fulminating disease should not have any surgery. I am sure they should have an ileostomy, and I am sure you can save lives in many of these cases.

There is one thing about the acute fulminating case, and it perhaps does not interest you as gastroenterologists; it is that you must not do a fancy ileostomy. These patients want something simple that will sidetrack their fecal stream, and that is a small incision, a loop of ileum put up and a tube put under it, and no more, with the idea that you will reconstruct the ileostomy later and save their lives. If you do a fancy ileostomy when they are in extremis, you will get a nice ileostomy but the patient will not be here to enjoy it.

SOME PERTINENT FACTORS IN CHOLELITHIASIS*†

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Many of us in this room are teachers, who are responsible to some extent, at least, for inculcating in our students a proper attitude toward, and treatment of the manifestations of disease as it affects the human body. Many of us are specialists or consultants, and as such are called in by general practitioners and family doctors to see their problem cases, furnishing us additional opportunity to extend our sphere of teaching to a postgraduate level on frequent occasions. For this reason it is of the utmost importance that we ourselves have a clear picture of all the ramifying implications and possibilities of any specific disease as it does, or at some future time may, affect the patient exhibiting it. Such ability is really our *raison d'être*, and without it we fail miserably.

One of the commoner diseases with which you, as gastroenterologists, have to deal is cholecystitis, and its usually concomitant cholelithiasis, and it is for that reason that I have chosen this subject for my address before your Association.

It would be impossible, even if desirable, to give any adequate review of the literature on the many aspects of surgery of the biliary tract, or merely of that portion concerned with the gallbladder alone. No such attempt will be made. Let us rather confine ourselves to the practical personal experiences of one reasonably active surgeon during his somewhat over twenty-five years of private practice, and see what conclusions may be drawn from such an approach. Let us further call on actual case records to present the picture to us; histories which deal with proven facts, uncolored by personal feelings or argumentative statements. First, however, let us generalize just a little bit, by way of introduction.

A few years ago there were published several papers, one or two read before this Association, I believe, on the subject of "The Silent Gallstone". It is true that at times cholelithiasis is discovered entirely unexpectedly, without any premonitory or diagnostic symptomatology, either during the work-up of a patient, or after death at the autopsy table. In the latter case one *might* call them "innocent gallstones", although even then who can say that in some instances they might not have been a contributory cause of death in some way, possibly as a focus of infection for an arthritis, an asthma, or a failing myocardium? But in regard to the living patient, who can look into the future sufficiently accurately to be assured that those stones will *remain* symptomless and innocent throughout the rest of that individual's life? And *if* they should later, even though unexpectedly, figure in some hazardous or disastrous episode in that patient's life, should he who originally withheld or failed to advise definitive treatment be absolved of blame?

During the past few years the two most popular topics for papers on surgery of the biliary tract have been those concerning either the extensive resection and

*Read before the Fourteenth Annual Convention of the National Gastroenterological Association, Boston, Mass., 24, 25, 26 October 1949.

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the complicated reconstruction involved in the attempted eradication of carcinoma of the ampulla of Vater or the head of the pancreas, or the equally difficult and often unsatisfactory attempts at reconstruction of the common duct, following previous injury of some sort. In the first instance, the ultimate *curative* value of this technically brilliant operation may be at least debatable, and it may even be questionable whether, in the long run and considering the operative hazards in the hands of any but a master surgeon, the average life span and intervening comfort of the patient is really increased over those results obtained by the much simpler, but acknowledgedly purely palliative procedure of cholecystgastrostomy or cholecystduodenostomy. In the second instance, the operation of reconstruction is made necessary by a previous surgical mishap in most cases, a fact which could be used as an argument against cholecystectomy, during the course of which most of these injuries to the common duct occur. With the improvement of postgraduate surgical training during the past decade, and the more universal recognition by most hospitals of the need for such training before acceptance of a candidate for their surgical staff, the incidence of such catastrophes should be materially lessened, and the validity of this objection correspondingly weakened. At the very worst, and considering the number of cholecystectomies performed under all conditions, these accidents are few and far between. In spite of the volume of recent literature on these two popular subjects, resection of the head of the pancreas and reconstruction of the common duct, between them they comprise a very small percentage of the surgery of the biliary system. Conversely, cholecystectomy is overwhelmingly the commonest of all biliary operations. For that reason it would not seem amiss to give a little time to its consideration. This is particularly apropos, since nowadays the trend of medicine is all toward the preventive side, and if it can be shown that a relatively simple operation, properly timed, can be used to thwart a possibly much more serious later eventuality, then we are merely keeping step with the modern times. There is much that can be said in favor of prophylactic medicine and surgery.

Technically this operation can be quite simple and easy, or it may, on occasion, be exceedingly complicated and difficult. The difference, by and large, rests on two factors, if one eliminates the physical characteristics of the individual patient. The first is whether the gallbladder is acutely inflamed or in a quiescent interval at the time of operation, and the second is how much intrinsic and surrounding damage has been done by previous acute attacks or chronic irritation before the patient has come to surgery. With both of these factors, the family doctor or the referring medical man is intimately concerned. True, there must always be a "first acute attack", and this *may* result in referral for, and performance of necessary surgery during the acute stage. Such, in my experience, would be a most unusual sequence of events, but entirely understandable, excusable and even commendatory. However, where previous warnings are disregarded, and symptoms not evaluated and investigated to determine the presence or absence of a causative cholelithiasis, or worse still, having determined the existence of stones and still not to have urged definitive action toward their removal, then when the

patient is faced with an additional complicating operative hazard, someone can be rightfully blamed for that fact. As long as stones are present in a gallbladder they are a potential menace to that individual's health and future well-being, and the sooner they are removed after discovery, the less chance is there for the development of complicating factors, to vitiate the good results which should be expected from their removal. These are the facts, and they *are* facts which we, you and I, have to impress upon our patients, our pupils, our professional confrères, and the public in general if we want to improve the results from, and minimize the hazards of surgery for cholelithiasis.

Lahey has expressed his feelings in no uncertain terms: "It is definitely my conviction that if patients were submitted to cholecystectomy before they had been through repeated attacks of gallstone colic, not only would the end results be infinitely better, but mortality markedly lower . . . Not only will early operation for gallstones be associated with a low mortality rate, but it will definitely preserve function and normal conditions in the biliary tract."

However, as a surgeon I have become all too familiar with the attitude of many a family physician or internist toward his patients as he watches them through subacute and recurring attacks of gallbladder trouble, or allays their fears when, unexpectedly, stones are found roentgenologically, or during the course of some unrelated operative procedure. Among the medical profession in general there seems to be a curious reluctance to advise or accept surgical therapy of cholelithiasis until the patient him- or herself precipitates the decision by an acute episode which does not subside under treatment with starvation, ice-caps, antispasmodics and opiates. This is most unfortunate, but possibly explicable on the grounds that each individual man does not observe many instances of the catastrophes which may result from too late resort to surgical measures, whereas the abdominal surgeon sees the accumulated group from many individual practices. Also possibly one of the reasons for late referral of cases to surgery is the poor results obtained *because* of such late referral, faulty logic, to be sure, but prejudice is notoriously unreasoning.

May I ask just one really pertinent question at this point? Is there any medical *cure*, not palliative treatment, of proven cholelithiasis? Of recent years there have been some rather interesting and even startling instances of the redissolving of renal calculi of certain sorts, in the living patient, by means of diet and medication, but personally I have never heard of any successful application of this principle to gallstones in the human patient. If and when such eradicatory treatment becomes possible, then, as a surgeon, I will gladly yield to my medical associates, but until then I will maintain that in almost every instance gallstones call for surgery.

If, then, the answer to that question is "No", and it can be shown that the presence of stones can be of real danger to the patient's future welfare, apart from any discomforts which they might or might not cause of themselves, is there not a valid corollary question which might be asked: "*Is* there an innocent gall-

stone, one which will not, at some future time subject that patient to an unwarranted hazard of health and life?"

That there is some risk to the patient in the operation of cholecystectomy is readily admitted, but that such risk is lessened when undertaken as a matter of choice in a silent interval, rather than as a necessity in an acute exacerbation, and before there have developed other complicating factors such as perforation, massive adhesions, diffuse cholangitis or hepatitis, pancreatitis or malignancy, to enumerate some of the more possible sequelae of medical temporizing, would seem to be equally obvious.

The following briefly abstracted cases, all drawn from my own practice, and most of them multiplied many times over, serve to illustrate and emphasize certain complications, some fatal but all serious, most of which I believe could have been eliminated by earlier surgery. In each instance the history, of long duration, is such as to leave little doubt of the diagnosis of cholelithiasis, although it had not always previously been made, and to give ample warning of and time to forestall just what eventually happened. Can we then hold medicine blameless when it did happen? Unfortunate incidents, yes, but largely preventable.

Case 1:—Mrs. R. L. Aged 51. This woman for several years had been having rather frequent "bilious attacks", characterized by digestive disturbances, moderate pain in the right epigastrium which would run through to her back between the shoulders, sometimes with nausea and vomiting, occasionally severe enough to require a hypodermic. Eight hours before her admission to the hospital she was "prostrated" by a sudden severe knife-like pain in the epigastrium, followed by nausea and vomiting. She was given two hypodermics and an enema, without affording her much relief, and then sent to the hospital about midnight, in moderate shock. When operated upon, the gallbladder was found moderately tense, thick-walled, and acutely inflamed, containing several stones; but the striking finding was diffuse fat necrosis, and a hemorrhagic, almost gangrenous appearing pancreas. No attempt was made to remove the gallbladder; it was opened, the stones and necrotic mucosa removed, and it and the common duct drained by catheters. There were no stones in the duct. The patient had a very stormy course, sloughing out a considerable amount of pancreas, requiring insulin to control the acute diabetes, and numerous transfusions. She was hospitalized for two months, and the wound was not entirely healed for an additional two months. A few years later she began having massive hemoptyses, and extensive esophageal varices could be demonstrated, without any evidence of hepatic cirrhosis. These, presumably, were dependent upon the scarring in the pancreatic area. She was a chronic invalid until her death from a profuse hemorrhage 10 years after her operation.

Acute hemorrhagic pancreatitis as an accompanying complication or sequela of chronic or acute gallbladder disease is not an unusual occurrence. The mortality rate in several series of collected cases varies from fifteen to over thirty per cent. Probably better than seventy per cent of these cases show cholelithiasis; it would seem therefore that stones *must* play some etiological role. The abdominal surgeon of reasonably wide experience sees two or three such cases each year, and dreads

them always. Many of them could have been avoided had previous warnings been heeded before the final acute episode precipitated surgical intervention.

While acute pancreatitis is a fulminatingly dramatic picture, much oftener the surgeon encounters, in his exploration accompanying the cholecystectomy, a moderate to severe induration and enlargement of the head, and less often of the body and tail, of the pancreas, which appears in the operative note as "evidence of chronic pancreatitis". How many of these already have or will develop sufficient fibrosis in the smaller intraglandular ducts, to interfere with their proper drainage and produce future discomfort for the patient, or even progress to diffuse pancreatic lithiasis or cystic degeneration?

Case 2:—Mr. J. W. Aged 56. This large, rather obese man had, over a period of five or six years, been having attacks of right upper quadrant pain, which would last for two or three days. There had been several episodes of mild jaundice of short duration. There was a great deal of chronic indigestion and flatulence. Three weeks before admission to the hospital he had a rather sudden excruciatingly severe pain in the epigastrium, running through to the back, which lasted for several hours in its acute phase. It was finally controlled by hypodermics. A few days later mild jaundice developed, which was still present when he entered the hospital. At operation a mass of old and fresh adhesions were encountered in the gallbladder area. The gallbladder itself was very thick-walled and enlarged, containing several hundred stones of all sizes. Fistulous openings were demonstrated between it and the stomach medially, the transverse colon inferiorly, and the duodenum posteriorly. These holes were all closed and the gallbladder removed, needless to say with much difficulty. No stones were found in the common duct; the head of the pancreas was quite indurated. After a very stormy convalescence of four weeks, this patient finally recovered and has been surprisingly well and comfortable, except for a rather large incisional hernia.

Case 3:—Mrs. R. B. Aged 70. This well preserved old lady had had attacks of pain in the right upper quadrant, radiating to the back and the right shoulder for "many years". They were usually fairly promptly relieved by a hypodermic administered by her family doctor who had told her that she "probably had gallbladder trouble", but made no suggestion that she have an operation, or even an x-ray examination. Thirty-six hours before admission to the hospital, she experienced "breath-taking" pain in the gallbladder area, and running through to the back, with marked nausea and almost continuous retching and vomiting. The pain rather rapidly came to involve the whole abdomen. She was given four hypodermics, an enema, two doses of salts and one of magnesium citrate, the latter three medications all being vomited. She was finally sent into the hospital after midnight with a clinically obvious general peritonitis, and a white blood count of 1,200! At operation, when the peritoneum was opened there was a flood of bile-like fluid containing large pieces of fibrin. There was a large perforation in the fundus of a big, thick walled, gangrenous gallbladder, which contained one stone the size of a hen's egg. The stone was removed, the gallbladder drained, but the

patient died 42 hours later of the peritonitis, a failing heart and pulmonary edema.

Case 4:—Mr. L. T. Aged 50. This, I think, is probably an unique case from several angles. Fifteen years before I first saw him, he was invalided for 3 to 4 months with a severe attack of pain in his right upper abdomen which his local doctor called cholecystitis and treated medically. Following this he had a lot of digestional difficulty, until 3 years before I saw him, when he had another bout of prostrating pain lasting several days. About one week previous to my being called, he began to pass tarry stools and feel rather weak. X-rays taken in the County hospital, where he was then admitted, were negative except for a calcified ovoid shadow the size of a large orange in the right upper abdominal quadrant. The blood in the stools became more red and more profuse and in spite of repeated transfusions, it became obvious that he was losing blood faster than it could be replaced! It was then that I was called up to the country to see him. Clearly something had to be done to try to stop the rapid loss of blood, which was thought from its character to be most likely coming from a Meckel's diverticulum. At operation the shadow seen in the x-rays proved to be a very large gallbladder, the lining of which was completely calcified to a thickness of 2 or 3 millimeters. There was a perforation between the fundus and the transverse colon, and caught edgewise in this ostium was a piece of the calcified lining, which had cut a rather large vessel in the wall of the bowel. The hole in the colon was repaired, and as much of the gallbladder removed as was possible. The patient did fairly well for awhile, but then I began to hear from his doctor that abscesses and sinuses were developing around the wound, several of which he incised. Eight months after the original operation, the patient was sent down to me, and it was found that he had an actinomycosis. There was a gradual decline over a period of 3 months, in spite of several operations and all manner of treatments. At autopsy he presented an actinomycosis of the abdominal wall, chest wall, right lung, diaphragm, liver and subhepatic area.

These cases illustrate results of perforation of the gallbladder. Some such perforations become walled off under the liver, some produce sinuses into adjacent hollow visci, most commonly the transverse colon, some fail to wall off at all, and precipitate a generalized bile peritonitis. Occasionally a perforation into the duodenum will allow the escape of a large stone into the bowel, with a subsequent intestinal obstruction necessitating operation. In every instance, the hazard to the patient and the difficulties of the surgeon are enhanced many fold. Unfortunately little emphasis is usually laid on the possible perforation of the gallbladder, in fact I have seen and heard statements that this rarely occurs. With such a viewpoint I would have to take serious exception, as in a relatively cursory examination of my own case records, I can uncover at least thirty such perforations of various kinds.

Case 5:—Mrs. W. B. Aged 67. This patient had been subject to frequent attacks of indigestion with pain in the upper abdomen for *over 20 years*. There was never any jaundice. About 7 months before admission to the hospital she had a

sudden attack of severe knife-like pain in the right upper abdominal quadrant, with radiation through to the back and up to the right shoulder, accompanied by nausea and vomiting. The acute attack subsided after two hypodermics, but ever since then she had some discomfort, although under constant medical observation and treatment. Five days before admission she had another exacerbation of pain, followed within 24 hours by increasing jaundice. She had lost 42 pounds in the 7 months. At operation the gallbladder was greatly thickened, containing one very large stone and several smaller ones. The common duct was moderately distended, containing mucoid mud and inspissated bile, but no real stone. The head of the pancreas was rather indurated. The gallbladder was removed and the common duct drained with a catheter. The pathological report was "chronic scarred gallbladder with acute exacerbation, and one small area of early primary adenocarcinoma near the fundus". This patient died of general abdominal carcinosis 25 months later, the longest postoperative survival in our series of primary carcinoma of the gallbladder.

Case 6:—Mrs. T. O. Aged 76. This rather obese old lady had been having so-called heart attacks for at least 15 years. They consisted of tight constricted feeling in the chest, smothery sensations, palpitation, gaseous eructations and occasional vomiting. No really thorough physical examination had ever been done; no electrocardiogram; no gallbladder x-ray series. She finally began gradually to get jaundiced, without any particular pain. Five weeks later she was admitted to the hospital. Though her icteric index was 48, her stools were never completely acholic. At operation there was a mass of adhesions under the liver, involving the omentum and the transverse colon. In the midst of this was a small, very hard, fibrotic gallbladder completely filled by one stone the size of a pigeon's egg. The common duct was much dilated and very firm. It was completely packed, clear back into the liver, with small stones and mud. One fair-sized stone was impacted in the ampulla. The pancreas showed much chronic fibrosis, and the liver chronic hepatitis. The common duct was cleared and drained, and the gallbladder removed with some difficulty; carcinoma was suspected. This was verified by the pathological report; in places it invaded the full thickness of the gallbladder wall. This patient made an excellent operative recovery, and for a year had no recurrence of the "heart attacks", but then went rapidly down hill and died 15 months after the operation, of carcinosis of the liver.

Case 7:—Mrs. E. T. Age 79. This patient had had her gallbladder drained and stones removed 20 years previously. Within 5 years of that operation typical gallstone colic began again, similar to that which she had before operation. These attacks had continued, 3 or 4 per year, throughout the intervening 15 years. About 2 months before admission she had a particularly severe attack which had never completely subsided, and during that time she had lost about 30 pounds. On examination, there was a long right rectus scar, the upper end of which was quite indurated, though not very tender. The liver edge was down a couple of inches, and felt rather irregular and bossed. This was quite tender. Diagnosis of inoperable carcinoma of the gallbladder, with liver and abdominal wall extension, was made,

but the patient's daughter, who was a graduate nurse, insisted on exploration; why she had not become concerned enough to force operation earlier is hard to understand. At operation, the preoperative diagnosis was verified. In addition there were several large stones in the gallbladder. A biopsy from a nodule in the liver showed primary adenocarcinoma of gallbladder origin. This patient died 2 months after the exploratory operation.

These cases are illustrative of different phases of primary carcinoma of the gallbladder, a subject which I reviewed in a paper read and published a few years ago. It would appear that somewhere around 4 to 5 per cent of calculous gallbladders will eventually undergo malignant degeneration, and once this has happened, the chance of cure by operation, or by any other means, is virtually nil. This alone, in the face of an average mortality from cholecystectomy of around 1.5 to 2 per cent, would justify early operation, disregarding *any* of the *other* complications which may attend gallbladder disease of long standing. I have now, in my own personal group of patients operated on since 1932, 21; since writing this, two others have been added, 23 cases of primary carcinoma of the gallbladder, all occurring in calculous gallbladders, most of which gave a history of fairly characteristic or easily recognizable preexisting trouble of from 2 to 20 years' duration. In 5 there had been a previous cholecystostomy for removal of stones, from 4 to 20 years before, with subsequent reformation of stones and development of malignancy, which in every instance invaded the abdominal wall, following outward along the old drain sinus scar. In almost every one of the 23 cases, a prompt cholecystectomy when trouble first began would have eliminated the possibility of the carcinomatous change which later occurred. At it was, when the malignancy had become established, every single case died of that malignancy, whether the gallbladder could be removed or not.

Acute hemorrhagic pancreatitis, perforation of the gallbladder and primary carcinoma of the gallbladder are all very dramatic and devastating complications of gallbladder disease. They almost always give a history of trouble existing long before the culminating episode which brings them to the surgeon. They almost never occur in an otherwise normal gallbladder, and must all, therefore, be considered etiologically dependent upon preexisting gallbladder disease, and in most instances stones. Because of these facts, are we justified in disregarding known cholelithiasis, or in minimizing to the patient or ourselves the warning of mild attacks or suggestive digestional disturbances, without thoroughly investigating their cause? *Can* we sit by, with a clear conscience, until nature and the patient *force* us into action by the acute crisis?

Now let us consider a smaller, and probably more controversial group of cases, where removal of a calculous gallbladder, whether from choice or because the patient forced it, produced unexpectedly good results in rather unusual circumstances.

Case 8:—Mr. T. G. Aged 49. In 1934 this patient was hospitalized for 2 months for what in every way, from his records, seems to have been a typical coronary occlusion of considerable size. Following that he had been having attacks

of pain in the precordial area, at times radiating to the back and left shoulder, but not down the arm, from 2 to 6 seizures a day and an occasional one at night. He had been living the very restricted life of a cardiac invalid for 5 years when he had a sudden increase in the severity of his supposed cardiac discomfort, together with marked flatulence, anorexia and heart-burn. His pain was relieved only by large doses of morphia. After 3 days, jaundice became noticeable, and 2 days later he was admitted to the hospital. The patient was highly nervous and most apprehensive. Positive findings were visible jaundice, moderate tenderness in the right epigastrium, a liver down about 2 finger breadths, relatively acholic stools, bile in the urine, icteric index of 53, white blood count of 12,450, and inverted T-wave in the electrocardiogram, blood pressure 140/95 and a nonvisualizing gallbladder. Four weeks after the onset of jaundice, this man was explored. The transverse colon was plastered up under the liver, around the gallbladder, and in a pocket between the two was some chronic granulation tissue and several small gallstones. The gallbladder itself was small and fibrotic, and contained several more stones. The common duct was explored but no stones found, and was drained through the stump of the cystic duct. The head of the pancreas was somewhat enlarged and indurated. The patient had no more of his anginoid pain following operation; his T-wave reverted to normal, in a few weeks he resumed his business activities after the lapse of 5 years—and also his week-end golf—and now, 10 years later is in excellent health.

Most of us have seen instances of the confusion of the symptomatology of cholelithiasis and coronary disease. The literature contains quite a few articles on the apparent close association of these two diseases, as well as of myocardial damage in combination with chronic gallbladder infection. It must be noted that this may be due to a basic underlying metabolic factor, common to both. Whether the patient just cited had actually suffered a coronary accident, in spite of the evidence of his record, might be debatable, but certainly he had had at some former time a perforation of the gallbladder, and equally certainly the cholecystectomy relieved his supposed anginoid symptoms immediately and permanently. We have seen this happen in several other instances:—in fact I have one such in the hospital at this writing, whose inverted T-wave has reverted to normal since operation. Apparently the electrocardiogram is, on occasion, no more infallible than the cholecystographic x-ray.

In the following group of cases, is it not fair to assume some degree of infectional allergy, in view of the results obtained by cholecystectomy?

Case 9:—Mrs. T. G. Aged 48 (a doctor's wife). This woman, over a period of 6 years, had become progressively more invalided by an asthma which no form of treatment alleviated. Otherwise she had no complaint of any sort until 3 days before admission to the hospital, when she was suddenly stricken with a typical severe gallstone colic. This progressed to the point where, in spite of the complicating asthma which would not permit her to lie flat in bed, it was felt operative intervention was imperative. At operation she had a tense, thick-walled, partially gangrenous gallbladder, filled with thick yellow-green pus and 60 to 70 facetted

stones about 1 centimeter in diameter. The common duct and pancreas were normal; a cholecystectomy was done. The asthma which we had feared as a postoperative complication, failed to materialize and she was able to resume her household duties and social activities in a manner previously impossible. This freedom from the former asthma remained until her death 5 years later from pneumonia.

Case 10:—Miss L. M. Aged 56. This saleswoman had been miserable with a generalized urticaria almost continually for 2 years, with several periods of acute incapacitating exacerbation. She had undergone complete allergy studies, but no definite cause for the trouble could be determined, nor any course of treatment successful. In the course of her studies, a gallbladder x-ray series showed previously unsuspected stones. At operation a moderately thick-walled gallbladder containing about 50 stones was removed. Immediately following this there was a severe flare-up of the urticaria for about 2 weeks, but she has been free of any further recurrence for about 4 years now.

Case 11:—Mr. A. J. Aged 51. For 10 to 12 years this man had been more and more crippled by a progressive arthritis of the combined rheumatoid-hypertrophic type. During those years he had been checked and rechecked at frequent intervals for possible foci of infection, resulting in the gradual removal of all teeth, tonsils, and 3 different operations on his sinuses. There was some history of indigestion, gaseous eructation and bloating, never with any significant pain, which was ascribed to inactivity induced by his arthritis. Finally, in one x-ray of his spine, faint shadows were seen in the gallbladder area, and administration of dye revealed, definitely, several stones. The *patient* insisted on an operation for their removal as being a possible focus of infection for his arthritis, though his *doctor* discouraged the idea. At operation a somewhat thickened gallbladder containing 8 moderate sized stones was removed. Culture of the bile grew a streptococcus viridans. This patient still has considerable crippling arthritis, but there has certainly not been the progression of the disease during the past 5 years which characterized its continuous course before the gallbladder was removed, and subjectively he insists that he has been much more comfortable.

In all of the foregoing cases the presence of gallbladder disease was totally unsuspected until found shortly before the operation. They had all been more or less incapacitated because of some apparently totally unallied complaint. And yet in each instance the patient was either completely relieved or markedly benefited by the removal of a chronically infected, calculous gallbladder. Possibly coincidence; possibly a "mental cure"; I doubt it as we have had other patients of this sort also. They at least suggest the advisability of investigating the status of the gallbladder when one is hunting for a possible "focus of infection", and removing it if it is incriminated.

Without further discussion, may I close this paper by repeating the question, "*Is* there a silent, an innocent, gallstone?"

Discussion

Dr. John M. McGowan (Boston, Mass.):—Dr. Finney is to be congratulated on this excellent presentation. From the files of his personal experience he has

presented cases which illustrate many aspects of gallbladder disease which are frequently overlooked.

The case which he presented concerning a patient with arthritis, asthma and cardiac degeneration illustrates a point which I have emphasized for some time, namely, that the nutritional disturbances associated with gallbladder disease often lead to a generalized degeneration of health and constitutional debility. This, I believe, is brought about in two ways:

First, the reflex irritation from the gallbladder itself produces disturbances of the digestive mechanism resulting in improper assimilation of food elements necessary to health; and

Secondly, infection from the gallbladder drains through the lymphatics into the liver directly and also along the veins which drain the gallbladder.

This results in the liver, so necessary to the elaboration of amino acids used in the repair of the body tissues and maintenance of good health, being impaired in its function.

Digestive disturbances, I believe, are due to impulses originating in the gallbladder itself, setting up a series of reflex disturbances of the digestive mechanism associated with antiperistalsis, pyloric spasm, or irritable colon. This reflex mechanism also produces disturbance in the blood vessels to the heart through the autonomic nervous system. There results an irritability in the cardiac mechanism which sometimes leads to paroxysmal tachycardia. In addition, evidence is accumulating to show that gallbladder disease may precipitate an incipient attack or coronary heart disease.

These presentations of Dr. Finney leave no doubt in our minds but that a patient who definitely has gallstones or a nonfunctioning gallbladder should be given the benefit of early surgery. If the patient is suffering a great deal of pain it is relatively easy to convince him of the necessity of surgery. It is evident to both patient and physician that surgery is necessary. However, if the patient is known to have gallstones or a nonfunctioning gallbladder, and is at the same time relatively asymptomatic, then the physician is faced with a different problem. He is required to give advice on the basis of future possibilities.

I have made it a rule that if a patient is under sixty and is in otherwise physically good condition, surgery should be advised. The patient should be given an accurate evaluation of the situation and an outline of the possible complications if surgery is not undertaken. Surgery should be advised on the following grounds: (1) rupture; (2) jaundice; (3) carcinoma; (4) nutritional disturbances.

(1) Rupture of the gallbladder occurs in the late sixties. A patient in the early fifties may hesitate to undergo surgery to prevent a complication expected fifteen years later; however, it should be pointed out that when a person reaches his late sixties, life will be found to be just as precious as it ever was, and he may not be in a condition to withstand the shock of a ruptured gallbladder.

(2) Jaundice: While at the Mayo Clinic in 1935, I collaborated on some research work on liver function in biliary disease and found that if the patient had more than one attack of obstructive jaundice, the liver was permanently

impaired in its function. It was, in most cases, capable of sustaining life, but the reserve was definitely and permanently reduced.

If there is a contraindication of the removal of the gallbladder, as a hypertension or a coronary heart disease of a serious nature, then one would consider leaving in a gallbladder which is not producing symptoms. Even a moderate degree of symptoms may be controlled by the judicious use of nitroglycerin, since a patient may be kept symptom-free on periodic nitroglycerin therapy, and in this way saved the suffering and discomfort of a surgical procedure which would not have added to longevity. On the other hand, even in the presence of a serious handicap such as cardiac disease, hypertension, or old age, the patient may be given the benefit of surgery provided he has sufficient symptoms, i.e., if he continues to suffer from gallbladder pain in spite of medical therapy.

We have a colored photograph of a gallbladder removed from a sixty-seven-year-old female, showing a perforation near the cystic duct. At the time of operation the patient was suffering from severe coronary heart disease. The gallbladder, however, would have quickly terminated her life span if not removed. The operation was done successfully in spite of her severe heart disease. On the other hand, as Dr. Finney has pointed out, it would have been much simpler for this patient if the gallbladder had been removed before the heart trouble developed.

Possibly one of the reasons why the internist is hesitant to refer his gallbladder patients early for surgery is because of cases in whom poor results were obtained. These poor results may be from complications and sequelae of the disease. These complications often are the result of not referring the case early enough in the disease, or may be due also to failure of the surgeon to bring to the operating table adequate knowledge of the physiology of the disease and system which he is treating. Some of the complications and sequelae are pancreatitis, recurrent cholangitis, and gastritis. One of our most valuable adjuncts in prevention of postcholecystectomy symptoms is an adequate exploration of the common bile duct and T-tube drainage. The common bile duct should be explored in approximately 50 per cent of all cases of gallbladder surgery. In each case a T-tube should be left in for prolonged drainage.

On the left of the next slide there is a lateral view of an x-ray of a common bile duct showing also the radicals of the biliary tree—the common duct is markedly kinked. On the right is an anterior-posterior view of the same cholangiogram in which this kinking is not obvious. This kinking occurs when the routine type of T-tube is used for common duct drainage. This tends to pull out and kink the common bile duct.

In order to prevent these complications we developed a tube at the Boston City Hospital, by using the principle of the inflatable bag.

Following surgery, on the fourteenth postoperative day *pressure studies* should be made and cholangiograms taken in order to be able to predict the duration of T-tube drainage. The T-tube should be left in a long time, if necessary. The resting intrabiliary pressure is an indication of the free flow of bile in the duodenum. Usually it should not be over 30 mm. of water above the ensiform.

Another intrabiliary pressure reading taken is the perfusion pain level in which pressure is elevated in the bile duct until the patient experiences pain. The pain pressure should be above 500 mm. of water. If it is below that we have evidence to show that the patient has cholangitis and is likely to suffer from continuous symptoms after the T-tube is removed.

We have three smoked drum tracings taken of intrabiliary pressure readings at weekly intervals. Only the beginning of the tracing is important in each instance. In the first—the beginning of the tracing has a pressure of 110 mm. of water, not relieved by amyl nitrite. Several weeks later the pressure begins at around 50 mm. of water, and finally, on the third, it begins at 10 mm. of water, indicating that the edema of the ampulla of Vater or the head of the pancreas has disappeared, and as far as this test is concerned, the T-tube may be removed.

Above the point of marked pain the pressure rises to 100 mm. of water. This pressure we produced by means of demerol. One hundred millimeters of water is a very low pain level and indicates, I believe, residual infection in the small radicals of the biliary tree, that is cholangitis, and therefore, the T-tube should be left in place for considerable time.

In an x-ray of the common bile duct and duodenum made by injecting each with a contrast media to show the physiology of the biliary tract with reference to the complications referred to you can visualize the common bile duct where it passes down behind the duodenum, and enters the duodenum at the ampulla of Vater. This patient had previously suffered from recurrent pancreatic edema and recurrent cholangitis. There was a moderate amount of duodenal spasm, and moderate narrowing of the lower end of the common bile duct, resulting in a back pressure in the biliary tract.

On the next slide you can see that we have reproduced by means of morphine sulphate what occurs during an attack of biliary colic. The lower end of the common duct is in a complete state of spasm; the ampulla of Vater is completely occluded. Such a mechanism would be conducive to back pressure in the biliary tract. Regurgitation of the duodenal juices into the stomach is noticeable, a mechanism which I believe produces a great deal of the gastritis in patients who have been allowed to suffer from gallbladder disease for many years.

In the completely relaxed duodenum following inhalation of amyl nitrite there is relaxation of the ampulla of Vater. There is very little regurgitation in the pancreatic duct.

Once again I want to congratulate Dr. Finney on his excellent presentation of this paper.

Dr. Howard A. Bouvé (Boston, Mass.):—I also want to congratulate Dr. Finney on his paper. I, of course, had the opportunity of reading it before this time. I enjoyed it immensely.

The facts that are to be considered in this type of surgery or in the diagnosis of gallstones, have been impressed on me more by the number of cases of carcinoma of the gallbladder I have seen in the last five years, occurring in the older people, in conjunction, often times, with acute cholecystitis, associated with cho-

lelithiasis. We have drained those cases, as we felt that they perhaps should not have a cholecystectomy, and at a later time we have found from the pathologist, from scrapings of the mucosa in removing the stone, that there was an associated malignancy. This is one fact I wish to leave with you, and Dr. Finney emphasized it also in his paper, that often these so-called "silent stones" are really dangerous and produce not only acute cholecystitis but also produce carcinoma by the irritation.

I believe that all gallbladders in these older people, associated with stones, should be removed for that reason, not only because of carcinoma, but also from the fact that a good many of these people in the seventies and eighties do develop acute cholecystitis, and if they develop stones in the common duct, it is certainly a much more dangerous procedure in the seventies and eighties than earlier. Most of those patients, you will find, have been to a doctor, and have been told that they had gallstones or perhaps a chronic gallbladder. They may or may not have been x-rayed. If they have been x-rayed and stones have shown, then they should have them removed, even if they are at the age where you would almost consider not doing surgery.

The other factor that I should like to suggest is that occasionally in acute gallbladder disease in the older people, when we find associated cardiac conditions or renal conditions, we do not dare remove the gallbladder. We simply drain those cases. I think you should scrape the mucosa and get a diagnosis from that. In some of them you will get carcinomas. In that case, at a later date, not too far distant, you should remove the gallbladder, because if they are allowed to run any length of time at all, they certainly have a very uncomfortable time and, of course, are all fatal.

Dr. Joseph H. Burnett (Boston, Mass.):—I don't think Dr. Finney would have any trouble selling this idea to a general surgical audience, especially some of the more recent graduates, because they are eager beavers on this subject; however, I discussed this with one internist and he disagrees, and another internist said, "There are too many operations for gallbladder disease, and I am sure that the general practitioner would not be sold on the idea."

I hope that Dr. Finney will see that this paper reaches these gentlemen so that they can read it and understand it, because I think that is where our trouble is coming from. The lay people still are of the opinion that gallstones can be dissolved, and occasionally one tells you about it. I am sorry to say I think some of the general men are of the same opinion.

On the service that I recently inherited at the Boston City Hospital, it has been the custom to take these cases that come in in the acute phase and with I.V.'s and penicillin try to get them to quiet down, and then operate on them at the end of two and a half to three weeks. We have used up hospital beds, but I am glad to say in the past year there have been no deaths under this régime.

I asked my resident to pick out the cases done in the last few months. He gave me a list of thirty-four. Twenty-three of them had many recurrent attacks; twenty-seven came in because they were in the acute phase, and at operation

thirty of the thirty-four showed stones in the gallbladder. The youngest was a young lady of twenty-two, and the oldest was a lady of seventy-four.

I should like to put one or two questions to Dr. Finney and get his reactions to the following: I operated in one case after forty-eight hours only to find in a sixty-two year old man, who weighed 218 pounds, a ruptured appendix with an abscess overshadowing the gallbladder on his liver.

Another case, a young lady of seventy-nine, with a history going back at least thirty years, of recurrent bouts of gallbladder disease, I operated, drained her gallbladder, took out a handful of stones—all of them, I believe—and the lady went on her way and six years later, at the age of eighty-five, I had to reoperate, drain her gallbladder, and I found one stone the size of a marble.

I should like again to thank Dr. Finney for the privilege of reading and discussing his paper, and I only hope that he can broadcast it a little bit farther than this audience.

Dr. Owen H. Wangensteen (Minneapolis, Minn.):—I have no profound convictions upon the subject. We must learn more about the formation of gallstones. Dr. Rewbridge published a paper a few years ago in which he indicated that stones demonstrable on a cholecystogram had disappeared following the administration of bile salts. It would have been better if Dr. Rewbridge had operated and shown that the stones previously demonstrated were absent following bile salt administration.

We know that stones form readily in the gallbladder of man and we know that in certain areas gallstones are prevalent. In Puerto Rico, for some reason or other, the incidence of gallstones among the population appears to be far greater than with us, a circumstance which suggests the possibility of an extrinsic cause. We need to divert some serious thought and study to such considerations.

The very formation of gallstones is a process we know very little about. Weiser (1933) has been able to initiate gallstone formation in vitro by pouring an alcoholic solution of cholesterol into a colloid binder such as gelatin or albumin and fibrin. Rhythmic circular banding occurs with the formation of "layered gallstones". This phenomenon is sometimes referred to as "Liesegang" rings.

It is well known, I believe, that when human gallstones are placed in the dog's gallbladder, they dissolve and disappear. Many of us have performed this experiment. By placing cotton and introducing an agar culture of a relatively avirulent organism into the gallbladder, I succeeded more than 20 years ago in producing in dogs a picture like chronic cholecystitis observed in man. The foreign bodies remained and formed the nidus for gallstone formation. These observations were never published. I have the impression that similar experiments have already been documented in surgical literature. Not being much of a biochemist, I abandoned the problem to pursue studies of bowel obstruction. There is of course a difference in the bile salt content of dog and human bile; that of the dog's bile is largely taurocholic bile salt; that of man glycocholic acid.

We know from studies of surgeons like those of John Berg, of Stockholm, that stasis has a good deal to do with the formation of stones; yet gallstones can form

within the common duct. If stones never formed within the common duct after excision of the gallbladder, I would like to go along with Dr. Finney's suggestion of excising the silent gallstone as a matter of routine. I agree largely with what he has said. My medical colleague, Dr. Cecil Watson, Professor of Medicine at the University of Minnesota, I think is far more enthusiastic about the excision of the silent gallstone than am I. He has a keener interest in and knowledge of diseases of the biliary tract than I. He is inclined to urge the excision of silent gallstones.

All that Dr. Finney has said about cancer of the gallbladder is very true. I don't know how many such fatal cancers are reported in the vital statistics of the United States each year. They probably are not many, but one way to prevent cancer of the gallbladder, as Dr. Finney has said, is to remove the gallbladder for silent gallstones.

One item that has deterred me from going along fully with Dr. Finney's suggestion is that I see at the University Hospitals in Minneapolis each year several patients with large incisional hernias and others with strictures of the common bile duct—all as a result of the excision of the gallbladder by persons eager to do surgery. Let no man throw stones in this discussion; however, let us look realistically at the problem. Rupture of the gallbladder, cholangitis, gallstone obstruction of the intestine as well as cancer of the gallbladder are all owing in part to a very conservative attitude toward the silent gallstone. Yet, it seems to me that, all these conditions, at least as I see them, are less frequent as a group than are the two complications of surgery of the biliary tract which I just mentioned: hernia and stricture.

While giving serious thought to Dr. Finney's recommendation concerning the silent gallstone, let us at the same time urge a greater interest in the study of gallstone formation. How much do extrinsic factors have to do with the formation of gallstones? Would perfusion of the common bile duct of man, through a T-tube, with heterologous bile salts help to dissolve stones which are not readily removed? The problem deserves study.

Dr. Samuel S. Berger (Cleveland, Ohio):—I wish to thank Dr. Finney for his excellent presentation.

I would like to have heard Dr. Finney discuss his method of dealing with the acutely inflammed gallbladder. Dr. Burnett made some pertinent remarks relative to this.

It has been my experience that the surgeons are more conservative than I am. As soon as the diagnosis of acute cholecystitis is made, the patient usually is ill only a few hours or a day, rarely longer. I urge surgical intervention immediately. I have found that surgeons are reluctant to operate in the presence of an acute infection. The dictum seems to be prevalent among surgeons that it is better and safer to wait for the gallbladder infection to subside before going ahead with the cholecystectomy.

I have never been able to foretell at the time when the patient is first seen whether the acute process will subside or progress to gangrene, perforation, empyema or liver abscess, etc. Nor have I met the surgeon who could assure me

that deliberate waiting, even under the most careful observation, might not lead to these serious complications. If the patient is first seen when the process is subsiding—the temperature, pulse, leucocytosis and local symptoms are disappearing—we are prone to wait but further delay may again result in a flare-up and serious complications. Not infrequently inflammation of the gallbladder is apparently of an allergic nature as observed in surgically removed material (B.S. Kline). Acute necrotizing lesions have been observed superimposed on subsiding inflammatory lesions in the gallbladder. This is an added reason for prompt surgical intervention even when the patient is seen late; that is, after 48 hours.

In the past I have seen these complications too often, many times at autopsy, to permit me to accept a waiting attitude. Certainly, in recent years, with more skillful technic, better anesthesia, antibiotics and better preoperative and postoperative care there is less and less reason for delay which, in my experience, is more dangerous than immediate surgery. I think the practice of waiting for the acute gallbladder to subside is still a too common practice both among surgeons as well as among medical men.

I should like to hear from Dr. Finney what his attitude is in the management of acute cholecystitis.

Dr. John M. T. Finney, Jr. (Baltimore, Md.):—I want to take this opportunity to thank all of the discussers, and I will attempt in a very few words, because we are late, to take up certain points which their discussions have raised.

I was much interested in the x-ray slides which Dr. McGowan showed—the angulation of the common duct, and the difficulties of the T-tube. We, in Baltimore—and I say that for most of the surgeons there—rarely use T-tubes for just that reason. Personally I don't believe I have used a T-tube for simple drainage of the common duct in ten years. Our preferable form is to use a small catheter, introduced, if possible, through the stump of the cystic duct after the removal of the gallbladder. If that is not possible, then to put it through the defect in the common duct, and suture it there with triple zero chromic gut, which will usually dissolve in about ten days, but in the meantime will insure the tube's staying in place. In that way a catheter, smooth, without angulation, will slide out when it is taken out, or when it comes out, without causing additional damage to the common duct, and we believe that that furnishes just as satisfactory a method of drainage with much less trauma and hurt to the patient.

In regard to the withholding of operation from the hypertensive and the coronary-diseased patient, I am afraid I would go just one step further than Dr. McGowan, and say that I believe that unless they are tremendously damaged and sick from those troubles, in at least certain instances their concomitant disease will be helped by the removal of an irritating gallbladder, and for that reason we do not withhold operation except in most unusual instances.

In regard to Dr. Bouvé's speaking of the old patients, my oldest cholecystectomy, which had to be done in a very acute stage, was ninety-one years old and survived. I have recently had a patient eighty-seven years old who had to be

operated on for a rupture of the gallbladder and she also survived, possibly because of the present knowledge of antibiotics. I am not one who feels that the mere matter of recorded years is a contraindication to operation; we see so many people at fifty who physically are older than other people at eighty, that I pay very little attention to the years as given when deciding on whether that person needs an operation, or, we hope, will benefit by one.

Dr. Burnett raised a question of the danger of operation in the acute stage. That is a very real danger, but not prohibitive.

Dr. Berger also asked that question. One of the reasons why the surgeon does not like to operate in the acute stage is not that the condition of the gallbladder itself necessarily is a contraindication, although the operation will be much bloodier and the gallbladder will be much more difficult to take out, but because of the tremendous surrounding edema which makes it much more possible to injure a cystic artery or to lose a cystic artery and injure the duct in trying to pick it up again, than in a patient whose gallbladder is not in the acute stage.

It is for that reason that the surgeon does not like to get a gallbladder in the acute stage after it has gone on for a matter of two or three days.

My own practice is this: If I can see that patient within thirty-six hours of the onset of that particular acute attack, then I make an emergency of that patient, just as I would of a suspected appendix—midnight, four o'clock in the morning, what not. If that patient has progressed beyond the thirty-six hour period, then I would prefer to allow an additional twenty-four hour period to see whether he is showing any evidence of cooling off, of subsidence of that attack, and, if so, allow the attack to subside—and I mean thoroughly subside. It will take probably three, four, five, sometimes six weeks before it has completely gone down, not symptomatologically, but as far as the gallbladder itself is concerned. If, however, after the additional twenty-four hour period, there is no evidence whatsoever of subsidence, then I feel one has to operate in order to avoid certain of the catastrophes having to do with gangrenous areas in the gallbladder and leaking through them.

Now, this is a dangerous statement to make, but most gallbladders will not perforate under about four days, so that your additional twenty-four hour period will give you time to watch if you have already overstepped your thirty-six hour period; however, as you know, there are exceptions to every statement, and one has to apply one's judgment to the individual case rather than try to make the cases fit one's accepted rules.

Dr. Wangensteen has raised certain questions which to me are vitally interesting, but, unfortunately, I am merely a clinical operating surgeon, and he is an investigator with the means at his disposal for those investigations. I would be eternally grateful and everlastingly interested if he and his coworkers, or somebody else, would discover why gallstones form. I know personally that I couldn't do it, because I am not equipped to do it. I merely have to accept them when they have formed, and for that reason I have confined the subject matter of my

remarks entirely to demonstrable gallstones. I purposely have avoided the somewhat controversial subject of cholecystitis without demonstrable stones. I further have avoided the question of various of the antecedents and sequelae of gallstone operations, such as the so-called liver dyskinesia and the upset in liver metabolism, and so forth, and so on. I certainly do not consider myself competent to talk on those subjects, but they are subjects of vital importance, and it is most important that somebody does investigate them and give us the final answer. Until that occurs, I am afraid I shall have to reiterate my statement that when you know there are gallstones there, the part of wisdom is to get rid of them, and I might add as corollary to that, that having done your job as a surgeon, to the best of your ability, then it is wise to turn that patient back to the internist to do his work in the follow-up, to try to preserve the normal functions, which are most necessary for that patient's future well-being.

PROBLEMS OF ACUTE INTESTINAL OBSTRUCTION*†

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Mr. Chairman, Ladies and Gentlemen: I must apologize for I have no manuscript. I was a little dismayed to find when the time rolled around that I should send a manuscript to the gentlemen who were good enough to consent to discuss my paper, that I had not written anything on the subject in four years; and yet in those years there have been no important developments pertinent to the practical aspects of bowel obstruction that are essentially new. I might say one other thing: In those early years of trial when I was a young professor at the University, it was customary, and it is even true, I believe, in the Boston area, that a new professor has to go out into the environs so that his colleagues can take his measure before full acceptance. The only subject I knew anything about was intestinal obstruction, and when Mrs. Wangensteen, on going home from one of these excursions, said to me, "Well, if you can't go next time and have no manuscript, I think I could give your speech for you," I thought it was about time I scurried around for a new topic. I confess freely I did wear the subject a bit thin on such occasions. But as I view it now, I presume I was searching the minds of others as well as my own for a new approach.

Well, in the intervening years I have remained essentially a plumber of the gastrointestinal canal. Occasionally, I have scampered up toward the top of it, and sometimes I have lent my efforts to the lower reaches of it, and yet my greater concern over this span of time has been with the alimentary tract as a whole; and the problem of intestinal obstruction is still a challenging one.

We can send Tom Thumbs with eyes and fingers for us down in the upper reaches of the alimentary tract and also into its lower reaches. I do not believe that all those maneuvers have yet been exhausted, and some of the developments which have come in intubation relate essentially to enlargement of that kind of vision. Every ploughboy who eventually comes to experience some success as an orator has probably made his most famous orations in the cornfield; at least, he had visions of making them. Every surgeon who stands long hours and operates, of necessity is occupied with the routine of cutting and sewing and tying ligatures on blood vessels; but unless his imagination can be stimulated to think of how his labors may bring greater promise of success to his patient, I do not believe that his labor is as satisfying to him as it might otherwise be.

Well, I must get to the problem that brought me here today. Sixty years ago, when the first Congress of Physicians and Surgeons met in this country to discuss

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in its broad perspective, among other topics, the bowel obstruction problem, Reginald Fitz, from the Boston area, participated, and after much discussion, it was generally agreed that after forty-eight hours of conservative treatment, the patient should be handed over to the surgeon for operation. The skills which the surgeon had to compete with in those days were, of course, employment of morphine, use of aperients, hot stupes, and colonic irrigations.

Today we debate which patient should be operated upon. There are those in our midst who suggest that every patient should be operated upon early; that bowel obstruction is essentially mechanical in its origin, and that relief of mechanical obstruction or occlusion necessitates ordinarily the employment of mechanical means. Yet our labors can be made less in certain instances by agencies which will deal successfully with the difficult problem of distention; and were it not for the sea of unruly gut which confronts the surgeon when he operates, I think more of us would capitulate willingly and wholeheartedly to the thesis that every patient should be operated upon the minute that he can be gotten ready for operation.

In 1889, when Sir Frederick Treves wrote the second edition of his book on bowel obstruction, he said that those who were enamored of statistics probably could no doubt show that it would be safer to jump from the Clifton suspension bridge than to have bowel obstruction and not submit to operation. Then, just a few years after the meeting of the Congress in this country, physicians and surgeons gradually turned toward admitting that patients should be operated upon as soon as the diagnosis was made; yet just twenty years ago now, in 1929, when C. Jeff Miller of New Orleans, discussed the experiences of the Charity Hospital Staff with the management of acute intestinal obstruction, he reported a mortality of 60 per cent. That accomplishment has been bettered considerably in the intervening years, and essentially, I think, through better understanding of the nature of the ill effects of bowel obstruction.

The Effects of Obstruction

Through taking advantage of conservative decompression measures which exceed in performance those recommended sixty years ago, re-enforced, in turn, by our better understanding of the nutritional, fluid and electrolyte needs of patients; and by accomplishing the synthesis of new information out of old facts—we are doing essentially the same operations for acute intestinal obstruction, using largely the technics of our forebears; however, we have perfected some of those technics so they can be used with greater precision today than in those earlier times.

In speaking of progress in our knowledge and understanding of bowel obstruction, I might restate just briefly this information: In 1912, Hartwell and Hogate, of New York City, gave saline solution to dogs with duodenal obstruction and found to their amazement they could prolong the lives of the dogs approximately three weeks. Haden and Orr later (1923) hypothecated that, the saline solution detoxified an unknown toxin, which they called X. It remained for Gamble and Ross, of this city, in 1925, to show that the ill effects of obstruction in the upper gut, were owing to the circumstance that the patient lost fluid and electrolytes, which, if adequately replaced, constituted good substitution therapy.

In the intervening years, we have learned to know something too of the effects of strangulating obstructions. Patients having this type of obstruction run the risk of loss of viability of the gut; and so too, in patients with sustained intraenteric pressure approximating the blood pressure at the venous end of the capillaries there is also a threatened loss of viability in the reaches of bowel just proximal to the obstruction. A sustained intraluminal pressure in the obstructed intestine prevents venous egress of blood, the gut wall softens and the protective barrier of the mucosa is lost, and in time noxious agents can permeate into the peritoneal cavity.

There was a time when it was taken for granted that in all obstructions there was increased absorption by the normal route, namely, the mesenteric portal system, but most experimentation has shown that distention per se reduces absorption through this area. It is viability of the gut wall that we are concerned with primarily in strangulating obstruction, and the relief of obstruction before that happens.

We know, too, that in strangulating obstructions there may be a large blood loss into the gut. The hurrying and quickening of the pulse in volvulus and intussusception are due essentially to the shock-like state which may accompany strangulating obstructions.

Well, when an appreciation of causative factors and when understanding and enlightenment come into any obscure subject, much good comes from that occurrence. That is one of the great benisons of research. It brings understanding and hope out of chaotic confusion.

Diagnosis

Now, with reference to diagnosis, we have made some progress. We are using essentially the criteria which our predecessors employed. We have come to appreciate more fully the uses of the x-ray film. The gaseous distention of the gut, especially if it is accompanied by evidences of *intestinal colic*, such as can be elicited by the stethoscope; the concomitant recurrence of intermittent, cramping pain, accompanied by loud intestinal noises—that occurrence suggests definitely the presence of *intestinal colic;* and if a patient has gaseous distention of the small intestine and has *intestinal colic*, he must have, of neecssity, some kind of bowel obstruction.

To confuse us, there are to be certain, such things as spastic ileus. On this score, I concede freely I have made so little progress with my ability to differentiate spastic ileus and mechanical obstruction that, within the past year, I have explored two patients with spastic ileus thinking they must have mechanical obstruction of the gut.

The escape of fluid into the peritoneal cavity in strangulating obstruction is essentially the agency which helps us to recognize the circumstance that the peritoneum is being irritated; and if, we think of the various grades of rigidity which accompany affections of the peritoneum, we know that the most rigid abdomen is the boardlike abdomen of peptic ulcer perforation. If one has once put his hand on such an abdomen, that experience exceeds the virtue of all description. It is like seeing an orange once; you recognize it readily when you see another. It is the irritating quality of hydrochloric acid which creates that rigidity. Contrast the lack of

such abdominal rigidity in perforations of the lower colon at the time of proctoscopy. The presence of feces in the peritoneum is fraught with great hazard, but the pH of feces is about the same as that of the blood. The patient may be totally unaware that anything untoward has happened and needs to be told of the mishap. Rigidity is therefore a reflection of the character of irritant in the peritoneal cavity. In strangulating obstructions, the peritoneum is stimulated by a mild irritant—blood escaping into the peritoneal cavity. Moreover, the parietal peritoneum is probably the most sensitive of all serous membranes, and if time permitted, I would like to digress on that matter, for I regard it as important in the recognition of acute abdominal disorders. There are of course opportunities for being wrong when the diagnosis of stangulating obstruction is posited largely on the item of whether or not there are signs of peritoneal irritation present. All of us who have employed this sign as a differential criterion have had the experience of operating and finding only a simple obstruction, accompanied by the exudation of fluid into the peritoneal cavity. That occurrence, of course, is not dismaying; it is very disconcerting, however, to find as time goes on that, the absence of signs of peritoneal irritation caused you to believe that the obstruction was simple in nature and, in consequence, a strangulating obstruction was overlooked. That may happen to anyone who affects to differentiate *simple* and *strangulating* obstructions.

In other words, the criteria which guide us in the differentiation are not absolute. Dr. Leland McKittrick of this area has stated that, he finds a polymorphonuclear leucocytosis the most helpful sign in this differentiation. My colleague Dr. Robert W. Toon is exploring this suggestion experimentally, contrasting the leucocytosis occurring in dogs following the establishment of simple and strangulating obstructions. A "U" suture can be placed about the gut and left in place until all effects of the abdominal incision have passed. Dr. Toon tells me, there may be, in the early hours following the establishment of strangulating obstructions, more patent evidences of leucocytosis; however, some hours after the onset, no clear cut difference is discernible.

Also, on this score, it should be said, there are occasions when patients with strangulating obstruction exhibit little or no demonstrable evidence of interference with the problem of intestinal transport; moreover, there may be neither fever nor leucocytosis. In other words, the differential diagnosis between *simple* and *strangulating* obstruction may be exceedingly difficult, if not impossible. In fact, one may debate whether obstruction is present at all.

In the main, the following are important in the recognition of intestinal obstruction:

Intestinal colic is present; that is, at the acme of intermittent, colicky pain, intestinal rumbling or borborygmi may be heard with the stethoscope. In colonic obstructions particularly, and in strangulating obstructions now and then, one may have to listen *long and often* to detect the presence of *intestinal colic*. Patients with obstruction of the small intestine ordinarily have feculent vomiting; and if the obstruction is of high grade, the vomiting may be frequent and copious. Strangely

enough, in obstructions of the colon, whereas distention may be great, there is frequently no vomiting.

In the old days one used to hear that patients with low obstruction have fecal vomiting. That only shows what poor observers physicians and surgeons may be. I have the feeling that for every ten people who can speak, there is only one person who can think, and for every ten people who can think, there is but one person who can observe, and persons who can both think and observe are rare indeed. Some of our difficulty on the score of observation is owing in part, I believe, to the circumstance that we find it so difficult to depart from what we are expected to observe; in other words, people who write textbooks frequently write of things of which they have no first hand knowledge. It is much easier to compose a book out of two or three others than to write one out of one's own experience; and it is only when one can delete from our textbooks these barnacles which have hung on through generations, and when people will write of their own experience, then we can begin to believe all that we read.

We are all at school in the University of Experience; its lessons are difficult and the assignments are long; its tuition fees are the highest of any school in the land. Every student and every teacher deserving of the name must ultimately be student and teacher in one person. He must learn to reflect upon the lessons learned in the University of Experience.

I repeat, it is in obstructions of the small intestine, not in low colic obstructions that feculent vomiting occurs; as a matter of fact, in high grade obstructions of the left colon, distention without vomiting is the rule; and when an indwelling tube is introduced into the stomach of such a patient, one gets a gastric or a bilious return; a stercoraceous vomit or aspirate through an indwelling tube suggests that the patient has an obstruction of the small intestine. Therefore, if the patient has *intestinal colic* and there is no abdominal tenderness; if the vomit or gastric aspirate is stercoraceous in character, you can be sure, long before you see the x-ray film, that this patient has an obstruction in the small intestine somewhere, and that it is probably simple or nonstrangulating in character.

The x-ray film is of the greatest help in telling *where* the obstruction is. Great distention limited to the colon in the presence of *intestinal colic* suggests definitely that the obstruction is in the colon. The lips of the ileocecal valve and the ileocecal sphincter make of this a closed intestinal loop, in the usual patient with colic obstruction. And in consequence, of course, the cecum may perforate. I have seen this occurrence in a thin napkin-like carcinomatous stricture of the ascending colon. If the distention is limited on the x-ray film to the small intestine, and there is *intestinal colic*, obviously the obstruction is in the small intestine. If there is distention of both large and small intestine, the x-ray findings would favor a diagnosis of nonmechanical ileus. In incomplete obstruction of the small intestine, there is ordinarily distention of coils of small intestine with demonstrable gas in the colon. And if, enemas are given to patients with such obstructions, the patient will continue to evacuate gas as long as swallowed air, the chief source of intestinal gas continues to slide past the point of obstruction. To be sure, there are atypical in-

stances of colic obstruction in which the lips of the ileocecal valve and sphincter fail to delimit the distention to the colon. In such instances usually, the inferior lip of the valve is deficient, thus permitting the small intestine to participate in the distention. This occurrence leads to confusion from an x-ray diagnostic standpoint, it being reasoned usually that, the patient has some type of reflex ileus. Ordinarily, the presence of *intestinal colic* helps to determine that mechanical obstruction is present; moreover, greater distention of the colon, together with a nonstercoraceous gastric aspirate, would decidedly favor obstruction of the colon with an incompetent ileocecal valve and sphincter. On the contrary, greater distention of long lengths of small intestine, accompanied by a stercoraceous vomit or gastric aspirate, favors strongly a primary obstruction of the small intestine.

I say all this here only to suggest that a careful integration of all the data is not only important but necessary to arrive at a likely diagnosis. The clinical and roentgen findings must be correlated with studied care. No clinician should accept with reservation, the interpretation of the x-ray findings as the final arbitrer of the situation. In all cases, a discussion of the findings is helpful to both clinician and roentgenologist.

To recapitulate, I believe it may be said that: 1. one can tell usually whether obstruction is present; 2. whether it is simple or strangulating in character; 3. whether it is complete or incomplete, and 4. whether it concerns the small or large bowel. You cannot tell with certainty what kind of obstruction is present. That usually remains a matter of conjecture, and those who advise early entry into the patient's abdomen in every obstruction may be fully justified in doing so on that basis alone.

Therapeutic Aids in Bowel Obstruction

Conservative decompression:—Wherein does the virtue of conservative decompression lie? Well, it resides essentially in the circumstance that the source of distention in obstruction is essentially swallowed air. Dr. McIver and his associates, twenty-three years ago, published from this area an interesting paper concerning the presence of swallowed air in the stomachs of patients after abdominal operations. Some years later, Dr. Charles Rea and I divided the esophagus of dogs in the neck. Shortly thereafter we obstructed the ileum. In order to obviate great distention of the obstructed intestine, we found it necessary to close the divided end of the cervical esophagus. Several dogs were maintained for relatively long periods of time in this manner, receiving only glucose and saline solution para-orally. The average survival period of 11 dogs treated in this manner was 35 days. A few dogs in the series received, in addition to glucose and saline solution, occasional (3 to 5) transfusions for 57 days. At autopsy, in several, there was no evidence of any gaseous distention of the bowel. Moreover, the quantity of fluid in the obstructed gut at death was small. In other words, the gastrointestinal secretions had been absorbed by this long closed loop of esophagus, stomach and small intestine following exclusion of the swallowed air factor. However, a few dogs still exhibited large quantities of both gas and fluid in the gut. This very circumstance we observe too in patients to whom suction is applied to indwelling tubes postoperatively to prevent intestinal

distention. In some patients, the method fails. When we learn more of the nature of this occurrence in the dog experiment listed above, we shall probably also understand similar occasional unpleasant and unexpected distentions occurring after abdominal operations in man too. We are again proposing to explore this occurrence further.

Obviously, the employment of suction in bowel obstruction may have certain ill side effects. It is possible without satisfactory checks to dechlorinate and dehydrate the patient. It is necessary therefore not alone to meet the fluid, electrolyte and protein needs of the patient; it's necessary too to cover the losses of Na and Cl ions through the agency of suction applied to long or short indwelling gastrointestinal tubes. Moreover, saline solution exhibits the virtues of a specific to replace fluids and electrolytes lost by vomiting only in very high obstructions such as are seen clinically in obstructions of the efferent loop of a gastrojejunal stoma. In obstructions of the lower reaches of the small intestine, or in colic obstructions, saline solution is in no sense the same protective agent which it is in high obstructions. In such instances, the primary problem is reduction of intraenteric pressure.

Oxygen inhalation:—Several years ago, Dr. Fine of this city, together with associates, proposed that the inhalation of high concentrations of oxygen be employed to displace the gas in the obstructed gut. The rationale of course is excellent.

Inasmuch as nitrogen as breathed is essentially inert and constitutes approximately 80 per cent of inhaled air, its exclusion in the inhaled gas, substituting for it oxygen which has a higher coefficient of diffusion, the nitrogen in the distended gut would gradually disappear. In our hands, at least, the method has not proved very practical. Suction applied to an indwelling tube is more direct and more effective. If an inert harmless gas with the same high coefficient of diffusion as CO_2 could be found as a substitute for the N_2 in the inhaled air, accompanied by an adequate amount of oxygen, the usefulness of this method in the relief of intestinal distention would undoubtedly be augmented considerably.

Inhalation of oxygen and helium at more than one atmosphere of pressure:—Another variant of this mode of attack, which merits some notice, is the possibility of combining a gas, like helium, which is not very soluble in fat or body fluids, with a proper amount of oxygen [75(He):25(O)]. If such a combination could be used at two or two and a half atmospheres of pressure, even for a few hours, the volume would be reduced considerably, thereby augmenting the opportunity for achieving ready decompression of the intestine by quicker descent of an indwelling tube. Even the problems of the surgeon, if operation is to be undertaken, would be simplified thereby. If the employment of more than one atmosphere of pressure does not complicate the picture further by creating the necessity for gradual reduction again to one atmosphere of pressure, the method might have something definite to offer. In any case, it would appear to be worthwhile to explore such an approach as an additional adjuvant aid in the bowel obstruction problem.

Helium and oxygen in this percentile relationship have been employed with considerable success in the rescue operations of divers. If the return to an atmos-

phere of pressure offered no important practical handicaps, certainly there might be much to be gained by the use of the method.

Indications for Suction

To which patients with bowel obstruction should suction be applied? Obviously, its employment as the sole agency of effecting decompression is contraindicated in all strangulating obstructions as well as in colic obstructions with great distention. This circumstance was recognized and emphasized when suction as an agent with which to combat mechanical bowel obstruction was described in 1931. In the intervening years, the Miller-Abbott and other long intestinal tubes have pyramided considerably the usefulness of gastroduodenal suction. The contraindications to its use as a sole agent of achieving decompression over prolonged intervals without simultaneous and a more aggressive surgical attack upon the obstruction remain essentially the same. Most of us, however, who are converts to or addicts of the tube in the treatment of distention, are a bit more conservative now than we were 15 to 20 years ago concerning continued employment of suction when the distention does not relent promptly. The real problem is still that of getting such long tubes to descend promptly to the lower reaches of the obstructed gut. If the tube could be gotten to the site of obstruction in 2 or 3 hours time, obviously the use of the method would be heightened considerably. The difficulties are many: the tube may not leave the stomach; when it takes 24 to 48 hours to get the tube well into the distended gut, it is apparent that conservative suction partakes of the shortcomings of "too little and too late" policies of operation.

Obviously something can still be done on this score. My associate, Dr. Grafton Smith, has constructed a tube by which he can control the tip by a removable and maneuverable obturator. In most instances he can get through the pylorus immediately (within 10 minutes) with this device. From there on, however, the descent of the tube, especially beyond the duodenojejunal angle may be slow. Procedures must be devised to accelerate the progress of the tube beyond this point. In postoperative distentions, the gut may be relatively inactive. Moreover, there may be an element of mechanical obstruction present to account for the distention, such that one hesitates to employ agencies such as pitressin or hypertonic saline solution to augment the contractile activity of the gut. The possibility of irritants injected into the gut through the tube, to stimulate the gut locally to increase the rapidity of descent of the tube, needs to be explored. My associate, Dr. John Wild has been working on an "intestinal creeper", which he envisions as migrating down the gut like a bullfrog hops along a tunnel. His device does just that over my desk, but when he puts it down a patient with great intestinal distention, the inertia occasioned by the heavy drag of the long tube, the impediment to its progress through coiled intestinal loops, almost fixed in position because of the rigidity created by the distention, and the very lack of adequate propulsive power of the gut itself—these agencies have served to nullify the thrust of Wild's device. In patients with slight distention, his "creeper" is more successful. It is in the patient with large distention, in whom the problems are the greatest, that we need the most help. And it is in

just such patients that conservative decompression is achieved almost invariably at a snail-like pace.

Operation:—In such patients, with known mechanical obstruction, obviously, earlier and more frequent consistent recourse to surgery should be had. McKittrick and others have been emphasizing the need for immediate surgery in all patients with mechanical obstruction. And were it not for the great problems presented by large and multiple coils of distended gut which eviscerate attending the incision in such cases, we would probably all capitulate readily to that point of view.

At operation, my associates and I have found that, employing Dr. Grafton Smith's device for getting the long indwelling tube readily beyond the duodenojejunal ligament, the surgeon's hand can often guide the tube with relative ease well into low-lying segments of distended bowel. This agency, therefore, permits ready accomplishment of operative aseptic decompression of the gut, an accomplishment which the surgeon must learn to cherish as a *sine qua non* of a successful operation for bowel obstruction with a uniformly low operative mortality. Elsewhere, I have stressed the necessity of careful adherence to this objective. Even antibiotics have not done away completely with the risks of spillage accepted by the surgeon for his patient when he operates for the relief of acute bowel obstruction in which intestinal soiling occurs at operation. Several years ago when a method of achieving the objective of relieving great distention without spillage at operation was presented before an assembly of surgeons, several well-known American surgeons came to the platform and said: "Wangensteen, you must have been speaking of leakage, not soiling or contamination."

"No," I retorted, "I mean spillage."

Despite antibiotics, I still regard avoidance of spillage at operation for acute obstruction as of great importance. Satisfactory release of the obstruction accomplished at operation with avoidance of spillage is the best assurrance that no undue license has been taken with the patient's life.

Operative aseptic decompressive suction enterotomy has practically done away with the need for blind enterostomy. Satisfactory relief of the distention makes management of the obstruction itself a simpler problem. Obstructive adhesions when present are to be divided. If the obstruction has been present for several days, (and this is one of the hazards too of prolonged conservative suction treatment), the adhesive band may have cut deeply into the gut. In fact, I have seen at operation the entire circumference of the obstructed bowel save its mesentery, divided by such a band, the edges of the gut being held together only loosely by fibrin. That such sites need to be handled and inspected with the greatest care needs no emphasis.

Strangulated gut must be excised. After decompression of the distended bowel, a primary end-to-end anastomosis can be accomplished ordinarily without difficulty.

Operation for The Obstructed Colon

The obstructed colon is a special problem. When long lengths of it are obstructed the great ease of decompressing it by transverse colostomy suggests it as

the operation of choice. When the distention has not been too great, and a long intestinal tube can be gotten into the cecum prior to operation, a primary resection is the operation of choice. For obstructions of the right half of the colon including the transverse colon, even in the presence of great distention, my associates and I have been doing primary resections quite regularly for some time, even though the descent of the inlying tube was arrested somewhere in the small intestine. The distended segment, together with the cancer and the potential lymphatic drainage area, is excised; and the undistended colon beyond the obstruction is anastomosed to the ileum end-to-end. If the ileum participates in the distention because of an incompetent ileocecal valve and sphincter, the distention of that segment is dealt with by one of the following methods: 1, employing the Grafton-Smith device for intubation, the long tube is threaded down into the lower reaches of the ileum and the cecum at operation, decompression being effected as the tube is drawn forward by the surgeon; 2, if this method is impractical for whatever reason, an aseptic decompressive suction enterotomy is done and 3, for lesser distentions, the segment of ileum, with which the colon is to be anastomosed, is evacuated by pushing the gas forward into the cecum or backward into more proximal reaches of the ileum, the contracted state of the gut being maintained by isolating the segment for the anastomosis between clamps applied lightly upon the bowel.

The majority of obstructions of the colon are simple obstructions. The opportunities for strangulating obstructions are not so numerous as they are in the longer lengths of the small intestine. The two primary strangulating obstructions seen not uncommonly in the colon are: volvulus of the cecum or sigmoid. In my experience, the former has occurred largely in postoperative patients. It is not too difficult to recognize. The clinical as well as roentgen picture is one of obstruction of the small intestine; in addition, there is usually a huge distention of the cecum which is pulled sharply upward and toward the left upper quadrant of the abdomen. There is also tenderness of the abdominal wall, especially in the lower right quadrant early. If one has any misgivings over the diagnosis, a barium enema should be done. A block in the colon is shown, which is owing to the circumstance that the hypermobile cecum and terminal ileum have undergone spontaneous torsion, the entwined coiling producing obstruction. The treatment is surgical excision and primary anastomosis, a procedure which has been carried out successfully many times in this clinic.

Many torsions of the sigmoid can be decompressed by the passage of a rectal tube as Bruusgaard has shown. Following the relief of distention by this maneuver, delayed or direct operation may be undertaken excising the long length of sigmoid colon which underwent torsion. My associate, Dr. Ivan Baronofsky, has carried out primary resection with end-to-end anastomosis directly following relief of the distention by proctoscopic intubation.

For the out and out colic obstruction with great distention involving the greater length of the colon, (owing usually to a cancer in the descending or sigmoid colon), performance of a temporary colostomy, upon the transverse colon through a transverse upper abdominal incision, (hence the designation transverse colostomy), is certainly the simplest and most effective manner of dealing with the distention. As

was remarked above, however, for all ordinary situations in the right colon in which the distended segment can be excised completely, a primary excision with immediate end-to-end union is carried out regularly in this clinic. Moveover, for even left-sided lesions with lesser grades of distention, the same type of operation has been done frequently by me and other surgeons in this clinic. Elsewhere, I have had occasion to say that, for cancers of the left side of the colon, a primary subtotal colectomy is the operation of choice, the ileum being anastomosed to the iliac colon at the sacral promontory. Recurrent rectal bleeding a short time after operation, from overlooked polyps or an additional cancer led me to adopt this policy, a practice which I have followed occasionally since 1944, and quite regularly since 1946, in dealing with all cancers of the left half of the colon. It rids the patient, at once, of a large potential polyp or cancer bearing area of mucosa. The procedure is followed only by transient diarrhea, in that, the entire ileum can be saved, unlike the situation in a primary cancer of the cecum, ascending colon or hepatic flexure, when the lymphatic drainage area contained in the vascular pedicle of the terminal lengths of the ileum must be excised. This latter operation excises the "water wringer" of the gut, in that the right half of the colon and a comparatively long length of the ileum are excised. (Incidentally, it is a *good* operation for obstinate constipation!)

I say all this here only to justify the present tendency in our clinic to treat an increasingly large number of patients with colic obstruction by primary resection. A primary *transverse* colostomy, more and more, is being reserved for the patient with great colic distention. In other words, at least in one area in which this practice is not mandatory as it is in the surgical relief of distentions of the small intestine, we are beginning to achieve the objective which should be the goal of all surgeons wrestling with the problem of bowel obstruction: to be able to contend with the primary cause of the obstruction as though the complication of great distention were not present.

Mortality

Surgeons have made progress in the bowel obstruction problem over the accomplishment of our ancestors largely because we stand on their shoulders and can see farther, not because our eyesight is any better. We must not be content, however, with existing mortality rates. They can be improved. Our objective should be eventually to deal with the primary cause of the obstruction as though the severe handicap of distention were not present. I grant freely, it is a lofty objective. Yet, when we learn to deal more effectively than we do now with the trying problem of distention—when we have learned to master that high hurdle, the objective outlined will lie within our reach. And when that time comes, the present serious threat to life of acute bowel obstruction will no longer appear as hazardous as it does now.

When Dr. Miller spoke in 1929, he reported a mortality in the Charity Hospital in New Orleans of 60 per cent. I don't know what the mortality is, by and large, in the country today. It is unquestionably far better than at that time. I have the impression that it runs in the area of approximately 20 to 25 per cent. In some clinics like our own, where a special interest is professed in the problem, the over-all mortality of acute obstruction is about 10 per cent. If we delete from consideration

the group of patients who come because of carcinosis of the peritoneum and those patients who presented intestinal distention complicated by peritonitis, the mortality in our clinic from acute intestinal obstruction can be said to be about 6 per cent. Yet for the same conditions, if the picture were not complicated by existing distention, the mortality would probably be 2 or 3 per cent. That accomplishment must be our objective for acute intestinal obstruction.

Summary and Conclusions

Distention makes the problem of successful management of bowel obstruction very difficult. The chief source of distention is swallowed air. Long sustained intraluminal tension may make a *simple* obstruction a potentially *strangulating* one. This occurrence is especially notable in the colon, where, because of the usual competence of ileocecal valve and sphincter, an obstruction in the colon beyond brings about a closed-loop, in consequence of which perforation may occur.

All therapy in obstruction, save decompressive agents, are aimed at improving the patient's condition. The limitation of employment of inlying gastrointestinal tubes to achieve relief of the obstruction, without operation, resides in part in the circumstance that: the method is not always effective; moreover, it is frequently so slow in achieving satisfactory decompression that, the equanimity of the surgeon and the safety of the patient are threatened thereby; (the nettling of the apparent serenity of the surgeon's temper is undoubtedly a good thing); but worst of all, occasionally the method may be inappropriately applied to patients with strangulating obstructions for whom immediate operation is always mandatory.

Nevertheless, suction applied to indwelling tubes has a role and an important one in the management of the distention of obstruction. And when we learn to devise satisfactory technics which will encourage quick descent of inlying tubes to the lower reaches of the distended gut, the usefulness of the method will be pyramided considerably.

If an inert gas with a coefficient of diffusion as great as that of carbon dioxide could be mixed with an adequate amount of oxygen to meet the respiratory needs, the inhalation method of augmenting disappearance of gas from the distended intestine would probably prove useful in the management of many types of intestinal distention.

Operation is still the mainstay of therapy in intestinal obstruction. Great distention is the *bête noir* of the surgeon in dealing with intestinal obstruction by operation. And when surgeons learn to deal satisfactorily at operation with this complication of obstruction, surgeons may some day hope to be able to treat the underlying cause of the obstruction as easily as though distention were not present. At the moment, that goal for many obstructions is beyond our reach; for some it has already been achieved. Continued work and thought upon the matter will help to make that hope and dream a reality. The surgery of acute intestinal obstruction is inching forward.

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Discussion

Dr. Jacob Fine (Boston, Mass.):—It is always a great pleasure to listen to Dr. Wangensteen. I have listened to him talk on intestinal obstruction before. He is direct and articulate, and always has something worthwhile to say.

There is little that I can add to what Dr. Wangensteen has said and written on the subject of intestinal obstruction. I would like to venture a few remarks that I believe will be helpful in the clinical management of this disorder.

If we are to avoid deaths, it is safer to assume that strangulation is present, in addition to obstruction, until it can be excluded with good reason. The existence of strangulation will be suggested by the presence of fever, an elevated white count, and a localized area of pain and deep tenderness. The pain is persistent, unrelenting, and not related in time or location to the pain of the colic. Colic is intermittent and radiates from the mid-line. The pain and tenderness due to strangulation is located where the peritoneal reaction is present, and the location of this reaction is where the involved loop lies. The clinical picture may resemble and be confused with that of a localized peritonitis. The distinction between these two conditions will rest in part upon the history, upon the x-ray evidence of generalized or limited distention of intestinal loops, and, if need be, upon the kind of fluid obtained by needle aspiration in the region of maximal involvement. The smear of the peritoneal fluid in strangulation will show a substantial number of red cells, while that of peritonitis will be almost entirely composed of leucocytes and bacteria.

Dr. Wangensteen's suggestion that the occasional absence of distention in strangulation obstruction may be due to intermittent patency of the lumen, strikes me as valid chiefly in the case of mesenteric vascular occlusion. More often such a situation will exist in an individual whose bowel proximal to the site of strangulation happened to be relatively empty when the strangulation occurred and who did not swallow food, liquids, or air in any quantity thereafter, or who vomited so much and so effectively as to virtually empty the proximal bowel. In these circumstances especially, the guides I have mentioned might prove crucial in the diagnosis.

Dr. Wangensteen has not found the use of 95 per cent oxygen for the treatment of distention helpful in intestinal obstruction. My colleagues and I recommended this method some fifteen years ago, not for intestinal obstruction, but for intractable distention, for which surgery, even if required, was not immediately applicable.

When the Miller-Abbott tube came into vogue for this purpose, we abandoned oxygen because it was distinctly inferior. That oxygen still has a place, however, is attested to by the surgical staff of the Children's Hospital, who, I understand, still use it in children when intubation cannot be performed. Moreover, several monographs in recent years from Scandinavia describe the successful application of oxygen inhalation in adults. Nevertheless, there is no doubt that intubation and suction constitute a far more effective decompressive procedure.

The advantage of operation over intubation and suction in obstruction of less than twenty-four hours' duration, which Dr. McKittrick has demonstrated, cannot be disputed. Nor will anyone dispute Dr. Wangensteen's warning of the dangers of prolonged intubation and suction, when it is not proving effective, or when it is too effective. If obstruction has been present for much more than 24 hours, surgical intervention is indicated as soon as decompression has been achieved. Continued suction may remove substances, such as potassium, which are not replaced by conventional fluid and electrolyte therapy alone. The recent introduction of potassium salts, in addition to water, sodium chloride, and blood or plasma, will presumably prove itself. None of these measures meet the need of restoring bowel continuity as soon as possible, so that oral feeding may be resumed and the continuing catabolic process interrupted or slowed down.

The increasing effectiveness of antibiotic therapy for intestinal disinfection promises to make a substantial contribution toward reducing the mortality from intestinal obstruction. Aureomycin with penicillin seems to be gaining ascendancy over other chemotherapeutic agents for effective sterilization of the bowel lumen, and particularly for control of peritonitis, which so frequently constitutes the final blow in intestinal obstruction.

In the experimental animal we have found that a strangulated bowel may survive long enough to permit a collateral circulation to be set up, if an effective antibiotic, applied locally, is given in time. The antibiotics preserve the integrity of the ischemic loop long enough to permit collateral circulation to be set up. This presumably results from (1) elimination of tension derived from gases generated locally by bacterial action on intestinal contents, and (2) by reduction of the inflammatory edema of the bowel wall produced by bacterial invasion.

The antibiotics have a place during the stage of unrelieved obstruction as well as during and after surgical intervention for its release. We are all familiar with rapid collapse and death which sometimes follows operative reduction of a strangulated hernia with replacement of loops of bowel, damaged but apparently still viable, into the peritoneal cavity. The accepted rule, that resection should be done rather than replacement of loops of questionable viability, may come to be modified as a result of more experience with antibiotics applied locally or parenterally in such circumstances.

Now I just want to add one more point in connection with antibiotics, because Dr. Wangensteen has sounded a note of warning that perhaps too much confidence is placed in antibiotics. I do not mean to imply, by suggesting more for them than he does, any reduction in the importance of observing the principles that he has so

well demonstrated. I am speaking in terms of the cumulative evidence of their increasing effectiveness. Material rapidly accumulating in experimental laboratories and in the clinic show what a remarkable antibiotic aureomycin is. You have all heard of Dr. Crile's recommendation that appendiceal peritonitis can be cured by penicillin alone. Rothenberg reports cures by pencillin alone of the peritonitis from gangrenous appendicitis in dogs. We have found that aureomycin is superior to penicillin for such a situation in the experimental animal. Bacteriologic studies on patients and on animals show aureomycin is the most rapid and effective sterilizing agent for the bowel that is available. It acts rapidly even when given orally and is at least as effective as penicillin.

Dr. Herbert G. Dunphy (Brookline, Mass.):—Before I received a copy of Dr. Wangensteen's paper on "Bowel Obstruction," as given today, I was consumed with curiosity as to what he might say on the subject. I took his book "Wangensteen, Intestinal Obstruction," from the shelf and re-read it to familiarize myself again with its contents. The book is well worth reading and re-reading. It ranks as a classic in its field. I recommend it to those of you who are interested in intestinal obstruction, and have not already read it.

Having perused the book, I was again curious to know how Dr. Wangensteen would condense an abundant store of information into the time allotted here. I find myself well pleased, as you must be, with his fine and scholarly presentation.

Duodenal suction drainage is an adjuvant to the therapy of all forms of intestinal obstruction. Dr. Wangensteen has pointed out to us that it must never be used as a substitute for definitive surgery in either large bowel obstructions or strangulating obstructions. Such errors have occurred in the past and will occur in the future unless we become more vigilant. Dr. Wangensteen recognizes that it is not always easy to differentiate between simple and strangulating obstructions. In old people particularly, who may have diminished sensitivity to pain and consequently diminished abdominal tenderness, and who may have a feeble leucocyte response to bowel injury, a strangulating obstruction may simulate a simple obstruction. Fortunately, this picture is not the usual one. Temporizing with suction drainage in such a case can be fatal. The possible occurrence of such an error plus the low mortality statistics from early operation, by which I mean obstruction of no more than twenty-four hours, or at most forty-eight hours, have influenced me to select operation in such an early obstruction.

The high mortality observed in late obstructions and in badly debilitated patients sometimes gives one a choice between suction drainage and surgery. One is often surprised by the unexpected recovery of a very sick patient offered only such suction drainage, intravenous fluids and blood and antibiotics.

Technical difficulties with suction drainage tubes are often encountered. The tube stays in the stomach in spite of skillful coaxing and proper directing with such aids as the fluoroscope. Gastric suction drainage is often adequate, but intestinal intubation is always more effective and often imperative to successful intestinal decompression. One can try the Miller-Abbott tube, weighted with mercury in the bag, or the mercury-weighted Harris tube. One can position the patient on his right

side to take advantage of gravity. One can use amyl nitrite to relax the pyloric sphincter. One can use the fluoroscope to direct the tube, but some duodenums still defy all attempts at intubation. I can recall much valuable time lost at such futile attempts and have resorted to enterostomy belatedly on more than one occasion because of such failures.

Need one emphasize here that one's responsibilities when using intestinal suction tubes, do not stop after placing the tube in the stomach? Too often its further progress is left in the hands of the inexperienced. Dr. Wangensteen has pointed out the advantage of having trained obstruction teams.

The percentage of failure in introducing suction tubes into the small intestine will depend on the degree of care and attention in following the course of the tube by experienced personnel. One will find frequent x-ray examination valuable both in positioning and following the tube and in determining whether the tube is accomplishing its mission. Is it relieving the harmful distention?

How long should one persist in the use of suction drainage in simple obstruction? One cannot indefinitely deny the patient the use of his gastrointestinal tract. Parenteral feeding has its limitations. If one does not have progressive relief of distention as evidenced by gross abdominal examination and better by x-ray examination to note distended intestinal loops, if one continues to have profuse enteric drainage from the suction tube, if one finds no gas below the suspected point of obstruction and no gas or feces by rectum, if one finds no evidence of absorption of fluids given by mouth with the duodenal tube shut off for an interval of three to four hours, and then opened, and then drainage recorded and compared to the intake—then one must assume that suction drainage has failed and early operative intervention is necessary.

If the patient not relieved by suction drainage is in poor condition, or if definite surgery is impossible for other reasons, Dr. Wangensteen believes in enterostomy. In this I concur. The temptation is usually too strong to do too much. There is not much satisfaction in doing too much, nor is there satisfaction in doing too little, too late.

Particularly in the postoperative patient who becomes distended after abdominal procedures and is not relieved by suction drainage, is decision that operation must be done to relieve an intestinal adhesion, difficult. I have recently watched such a patient for eleven days before operating. It was five additional days after the secondary operation before my initially established transverse colostomy drained.

The problem is not simple. We are indebted to Dr. Wangensteen for clarification of many of the poorly understood details of pathological physiology encountered in obstructions. I know he will continue to be a leader in advances to come in this field of gastroenterology.

Dr. Stanley J. G. Nowak (Boston, Mass.):—As a discusser of Dr. Wangensteen's comprehensive and authoritative paper on "Intestinal Obstruction", I should like to take this opportunity to add several items to the ever-widening scope of this important field.

The first item I wish to describe is a new, but admittedly rare, symptom of subacute mechanical obstruction, namely *back pain* located over the dorsolumbar region. I have observed this symptom in two cases of subacute mechanical obstruction. In one case my advice to operate for partial mechanical obstruction of the small bowel was not accepted until several weeks later when an acute shutdown occurred at which time a benign leiomyoma of the terminal ileum was found as the obstructive cause and was successfully resected. In the second case the symptom of back pain misled the attending physician into the belief that he was dealing with intercostal neuralgia. Approximately a fortnight after the onset of this and other better known symptoms of partial intestinal obstruction, exploration revealed terminal ileal obstruction due to adhesions. It is obvious how a symptom referred to the dorsolumbar spine might lead one astray from the diagnostic possibility of intestinal obstruction. I presume that the mechanism of this referred back pain is of the axone-reflex type with overspilling of afferent impulses from the celiac ganglion into the dorsolumbar spinal nerves.

The second new sign relates to the condition which I should like to designate as partial internal wound dehiscence. For many years Dr. Irving J. Walker of Boston has stressed the telltale significance of serous and serosanguineous exudate appearing on the dressing of an otherwise clean abdominal wound or on about the fifth or seventh postoperative day as a sign of impending eruptive dehiscence. I should like to call your attention to this same manifestation which occurs in a gradual dehiscing wound into which has slipped a loop of small bowel. This type of dehiscence occurred in a middle-aged obese man following appendectomy for gangrenous perforated appendicitis. This patient's appendix was removed through a right rectus muscle-splitting incision. His convalescence was characterized by fresh bleeding from the wound forty-eight hours after the operation, which was associated with vomiting and abdominal distention which was masked by his marked obesity. He was treated by intubation with prompt temporary relief but recurrence of distention on the fifth postoperative day with persistence of slight hemorrhagic discharge from the operative incision. When seen by me on the sixth postoperative day, there was moderate fever, normal blood pressure, clinical appearance of toxicity, rather marked distention with obesity, occasional bursts of hyperperistalsis, apparently clean wound, with a slight amount of serosanguineous discharge on the dressing. Rectal examination was negative. Roentgenograms showed marked dilation of the small bowel with some air in the colon. There was moderate leucocytosis of 11,700 with a slight left Schilling shift in the differential count.

Biochemical studies on this patient revealed the presence of a third infrequently observed condition, namely prerenal azotemia. This patient's NPN was 190 mg. per cent, serum chlorides 76 M.E.Q.

It was obvious that any operative interference at this stage would be hazardous. Harris tube decompression was instituted with electrolyte correction over a period of one week with continued vigilance directed toward the appendectomy wound. On the fourteenth postoperative day and seventh day of azotemic treatment with the NPN level 55 mg. per cent, this patient presented a complete de-

hiscence. Within several hours the dehiscence was repaired under spinal anesthesia, at which time a knuckle of the terminal ileum was found glued to the lower angle of the split rectus muscles. This patient made an uneventful recovery.

Prerenal or extrarenal azotemia was observed by Froin and Marie in 1912 in choleriform enteritis. It was described as a complication of intestinal obstruction by Tileston and Comfort in 1914. These and subsequent writers have described the clinical factors of this condition as one of renal failure without actual renal disease, with elevation of the nonprotein nitrogen blood level, hypochloremia, oliguria, or anuria, normal blood pressure or hypotension, with nonremarkable urinary findings. Various mechanisms are invoked in the causation in this syndrome, including impaired elimination of nonprotein nitrogenous products, increased production of these products, diminished renal blood flow with or without hypotension, etc. These syndromes require emphasis in any discussion of intestinal obstruction.

The third item I wish to bring out is the observation of chyloperitoneum of the true chylous type in a patient with acute mechanical obstruction of the terminal jejunum at the site of a Gilliam suspension. This patient was operated upon five hours after the onset of symptoms. The patient denied having any fatty foods during her preceding meal, four hours before the onset of her symptoms. At operation this patient presented incarceration of a loop of terminal jejunum measuring approximately 8 inches in length with its mesentery and distended lacteals. On opening the peritoneum, the first presenting abnormality was the presence of approximately 700 c.c. of pink-stained chylous fluid. This patient's obstruction was easily relieved, and convalescence was uneventful.

The final original item I should like to bring out is an observation related to adynamic ileus produced by mesenteric embolism. I wish to stress that the presence of bloody or tarry stools need not indicate a hopeless stage of strangulation in this condition. This sign has been observed by me in a patient who had incomplete superior mesenteric occlusion by an embolus of forty-eight hours' duration with frankly bloody stools during this period. At operation he manifested an entirely normal-looking terminal ileum and ascending colon to most of my colleagues in spite of my insistence upon the presence of ischemia which was supported by the absence of pulsation in the intestinal arcades. Resection was waived. This patient proceeded to show progression of this disease, confirmed at autopsy. From these observations it might be concluded that bloody or tarry stools observed in mesenteric embolism are due to a diffuse intestinal diapedesis with massive capillary hemorrhage into the intestinal lumen and not necessarily to a gangrenous state.

To these remarks dealing with perhaps rarely observed or rarely described symptoms and signs, I should like to add my support to the school of thought which stresses early surgical treatment whenever possible with the well-known exceptions in mind. Another way of putting this concept is to emphasize the fact that in dealing with intestinal obstruction *it is later than one thinks. If one is suspicious of the presence of this condition, it is very likely already there; and when one is reasonably sure that the condition is present, it is quite likely too late in the game.*

Dr. Earl J. Halligan (Jersey City, N. J.):—I should like to ask Dr. Wangensteen a question: How frequently does he perform enterostomy, and what are his indications?

Dr. Wangensteen has mentioned the problem of spastic ileus. That sometimes has been a nightmare. I remember a case before we had the Miller-Abbott tube, or Levine tube, or any type of suction, a girl about twenty-eight years of age, admitted with intestinal obstruction. On exploratory operation all we found was a spastic loop of ileum. We did an enterostomy above the loop. One week later she developed another area of spastic ileus about three feet above the original loop. We again went in and did another enterostomy, finding no other pathology. Two weeks later she developed one higher up, and we again did another enterostomy. By this time we were tired of doing enterostomies. She died.

We had a very careful autopsy and we could find no pathology. She had a definite spastic ileus about two or three inches in each loop. Each time it occurred, it was higher. We never did find any pathology. Even the spinal cord was taken out and examined for the possibility of some type of irritation. We never did find out what caused it.

I wonder if Dr. Wangensteen could help to illuminate that situation. Through the years I have often wondered what it was that happened to her.

Dr. Owen H. Wangensteen (Minneapolis, Minn.):—I am very grateful to you for the honor implied in asking me to come here and participate with other distinguished guests in this program. I always feel when I go home from a meeting such as this, that I have learned a lot more than I have brought to the meeting, and the very concentration of some thought, of course, upon discussions like this, is bound to enliven one's imagination and give one new perspectives.

I think it is quite apparent from the discussion that surgeons are today employing earlier operative intervention than we did, let us say, fifteen years ago. In 1888, when the first Congress of Physicians and Surgeons met in this country, it was decided that one should wait two days before having recourse to operation; by the early '90's it had been generally conceded that operation should be done immediately the moment the diagnosis was made; yet as late as 1929, in following out that practice, the mortality was exceedingly high.

Then we came into an era in which intestinal intubation had its vogue; in time, with increasing experience some of the limitations of the method were learned and gradually most of us settled down to our respective points of view with reference to what one might reasonably expect from suction in the management of obstruction; some amongst us have on occasions, expected too much; and when one does that, one is likely, of course, to encounter disappointment. There are those who continue to believe that, suction plays only a minor role in the treatment of intestinal obstruction and that all patients should be operated upon as quickly as possible.

I think the advocates of this latter policy of immediate operation in all cases should come forward with data to substantiate the validity of their thesis. Moreover, they should be able to indicate that their practice in their own clinics outstrips

the accomplishment of those who affect to integrate conservative decompression as an agency of therapy into the surgical management of acute obstructions.

We all like to think, of course, of utopian eras in which we deal with good-risk patients; of patients whose obstructions are not too serious; and that the item of distention itself would offer us no real problem; and that we could manage the most difficult obstructions without committing any errors of judgment or of execution. To ask a patient to withstand the possibilities for errors of commission as well as omission on our part, whatever they may be: of judgment, of execution; and to support his illness in addition, is sometimes asking a great deal of the patient. Yes, we are very fallible.

However utopian we would like things to be, we surgeons must be practical folk; we have to deal with situations such as they are; I must admit that I wrestle with the problem of acute bowel obstruction in our clinic less often now than I did in earlier times—and yet, I must admit even in operating on early cases presenting considerable distention, the problem of the unruly sea of gut is a disturbing one; if advantage of preliminary decompression can be taken, it is indeed a helpful adjuvant measure.

Now, I can think, as I look at the broad perspective of the problem that we might be able to get tubes down into the lower reaches of the distended small gut in two or three hours; that this interval of time will not absorb the complete attention of the patient; in other words, during this time, he can be given intravenous fluids and be gotten ready generally while we are making preparations for operation. I would like to believe one could guide the tube before operation into the jejunum beyond the duodenojejunal angle. Now, if the tube does not migrate quickly down the bowel, permitting ready decompression, early recourse probably should be had to operation. It is with reference to what commitment is made at this juncture, in event immediate decompression is not achieved, that differences of opinion would arise. Some would say: Wait longer; decompression may yet be achieved. Others would say: No, we cannot wait longer; we must operate at once. For this latter group, the Grafton-Smith device for securing intubation of the upper reaches of the jejunum will prove a great boon, as well as for achieving a larger number of satisfactory decompressions in certain types of obstruction without operation.

When the incision is made, the surgeon can locate the balloon-tipped catheter lying near the origin of the ligament of Treitz, and without much difficulty usually he can achieve decompression of the distended gut as he guides the inlying tube into the distended segments of gut near the obstruction.

We are doing many more primary operations now than in earlier years taking care of the situation ideally when operation is undertaken for the relief of distention. I want to emphasize again the great difficulty which confronts the surgeon in dealing with the problem of distention at operation. Were it not for the problem of distention, the mortality of acute bowel obstruction would be minimal; the division of adhesions and the repair of a hernia are not operations which command a large mortality. It is what these conditions might do to the gut that is important, and

what that occurrence does to the person, which makes intestinal obstruction and the distention accompanying it a difficult problem to deal with. When we surgeons learn how to combat distention at operation without spillage and when our success in achieving decompression operatively rivals that of suction treatment in those cases in which that treatment succeeds, we will have some occasion for complimenting ourselves upon our surgical skill.

Antibiotics obviously have some virtue in obstruction. One of the difficulties which hedges about their employment in surgery generally is that the continued administration of antibiotics may hide the development of an abscess. We have all seen this; it is a circumstance to which a surgeon must learn to be more alert. I shall tell you briefly about a recent painful experience. In March of this year (1949), I did a total gastrectomy and excised half the pancreas for a man in his sixties for a very large gastric cancer; about a week later the patient was dismissed and went home. His physician called me about five months later and said, "Our friend has a bowel obstruction". I was busy and my beds were full, and the getting of another patient into the hospital was not easy. I said, "Why don't you put a catheter down? Maybe that will take care of the retention".

A week later, the patient came into our hospital in the evening. He did not appear to be particularly ill and no one said anything to me about it. As I made ward rounds before going to the operating room, the next morning, I looked at the patient. He was up shaving though he had a nasal catheter in the intestine. I examined him carefully. He had definite abdominal tenderness; there was a rectal mass and slight distention of the intestine; and obviously the patient had a strangulating obstruction. At operation, the sight that confronted us startled me. He had several months before lost his stomach and a good portion of the pancreas. He had gangrene of somewhat more than half of the small intestine and of the colon up to its midtransverse portion. In addition, there was a gangrenous segment approximately 40 cm. in length just beyond the duodenojejunal angle. It looked like a hopeless situation. The gangrenous gut was very much like a fragile black ribbon; it hadn't perforated; there was, however, foul fluid in the peritoneal cavity. Prior to operation, we had noted on the x-ray film taken, a short thin rim of gas outside of the wall of a segment of gut. Obviously, there was peritonitis but there was yet no demonstrable leak or actual perforation. I did two intestinal resections; a short segment was excised at the duodenojejunal angle and the distal half of the small intestine and right half of the colon were excised, oblique end-to-end anastomosis being made at both sites of resection. Antibiotics were placed in the peritoneal cavity and were administered also postoperatively. The patient again got well and went home. A few days later, he came back with the story that he had had a convulsion. I didn't examine him too carefully, thinking the jig was up; one of my junior associates, Dr. Kirulik, said to me: "I believe that man might have an intraperitoneal abscess". I didn't heed that suggestion carefully enough. A couple of days later, the patient died; there was a silent right paracolic abscess in the right lateral gutter.

The loss of my friend, Thompson, who had survived two aggressive surgical assaults at my hands taught me an important lesson. Directly thereafter, I formulated in my mind a mode of attack upon such problems. And in a clinic, such as ours which busies itself daily with problems of formidable surgery upon the alimentary tract, we did not have long to wait. It was quite clear to me that: Mr. Thompson had peritonitis at operation; that the administration of generous amounts of antibiotics had masked the developing abscess; that an abscess needs surgical drainage. I had several long suckers with 4 holes at the tip made up (tonsil suckers) attached to long handles with an air vent over the mid-portion of the shaft which could be sealed off intermittently with the surgeon's thumb when the suction was in force. My plan was to explore the peritoneal cavity through 4 tiny incisions (if necessary). The first incision is made in the area under most suspicion. When 4 incisions are made, their location is as follows: one under each costal margin, just under the tip of the tenth rib in the nipple line; the other two are similar short incisions so placed on either side of the abdomen that the lateral paracolic gutter spaces as well as the cul de sac may be drained. If an abscess is encountered with the first incision the other sites are not drained. I feel that I can examine the entire peritoneal cavity quite satisfactorily by this method. The incisions are small; if pus is encountered, a stab wound is made for the exit of the drains and the incision is closed with interrupted sutures. If a large abscess is encountered, an air-vent suction drain of the sump variety is inserted as well as drains of the Penrose type. The former are removed usually after approximately 48 to 72 hours. When drainage is wholly dependent upon the Penrose drains, obviously it becomes necessary to have the patient lie flat on his abdomen several times a day for 20 to 30 minutes at a time, in order to get the aid of the force of gravity in augmenting drainage.

The subphrenic, supraphrenic, paracolic and cul de sac areas can be explored in this manner. It has been the agency of saving several lives since it was first used in our clinic, following Thompson's death several months ago. Yes, we have had a few negative explorations; but what is important is that we have saved lives by recognizing the presence of occult intraperitoneal abscesses; in addition, we have shortened convalescence in other patients.

To be sure, the method departs from the teaching of convention on this score, in that the abscess is not opened *extraserously*. I regard it as far more important however to *find* the abscess. Moreover, I have seen no suggestion that any harm has come to any patient through its employment. I have chosen to call it the suction-tip method of exploration for occult intraperitoneal abscess. I have the feeling that surgeons have not been as alert to the occurrences of this complication after operation as we should be. In our hands, it has been a very helpful expedient. Most of us, who go to the autopsy room to ascertain what defeated us in some of our endeavors, can recall instances like that of Mr. Thompson, in which finding the abscess would have saved the day. Suction-tip exploration for occult intraperitoneal abscess will help to make that occurrence a surgical curiosity.

Dr. Fine's discussion interested me very much; there is no doubt that if antibiotics could be taken orally by every patient likely to have an obstruction before

it occurred (sulfasuxidine, streptomycin and aureomycin) that when the patient got his obstruction the bowel would be more sterile and the problem wouldn't be so great. Moreover, as in the instance of Thompson related above, paraoral administration of antibiotics helps. It is not quite the same thing, however, as getting there with the drug before the obstruction occurred. Let us grant freely that the liberal use of antibiotics extends the accomplishment of surgeons in difficult surgery; that it does too in the surgery of bowel obstruction. I do not believe that I am far from wrong in suggesting that probably most of us here in our daily work give antibiotics more liberally than the *specific* accomplishment thereby justifies. We want to leave nothing undone. Yes, I follow along in that attitude. Let me say also, however, that, in colic resection, I give no antibiotic prior to operation. It is my feeling, and our experience bears that suggestion out: a precise, well performed resection in which there is no suggestion of spillage, employing the closed anastomosis, is the best assurance against infection.

My associate, Dr. Arnold Kremen has been restudying the problem of strangulating obstruction in our laboratory. Ligature of the portal vein in a dog causes the animal to die of hemorrhage into the gut within a couple of hours, an observation made in our laboratory about 20 years ago. Dr. Kremen finds now that almost uniform failure in having such dogs survive can be supplanted by a high incidence of success by the administration of heparin. That accomplishment has some importance for us in the consideration of a condition such as mesenteric thrombosis. If a segment of gut has no blood supply, the administration of antibiotics will only put off the time when that segment of gut will necrose. If the channels of flow can be kept open, there may be some opportunity for survival of that segment.

My misgivings over continued employment of suction applied to an indwelling tube, when satisfactory decompression is not readily achieved, are occasioned by the circumstance that so much is left to chance. Yes, the simultaneous administration of antibiotics and heparin might help to keep a potential strangulated loop alive. I would much prefer, however, to resolve the problem by having a look in to see what is going on. In patients with dubiously viable bowel, we inject 5 c.c. of a 20 per cent solution of fluorescein through the rubber tubing through which the intravenous fluid is running in. The lights in the operating room are turned off and with the aid of a Wood's lamp, the viability of the segment of gut is determined readily; if it remains purple in color, obviously it is not viable; if however, a yellowish sheen becomes apparent upon the surface of the strangulated segment, one may be certain it is viable. I have the feeling that we are excising, on less indication than previously, dubiously viable segments of gut employing this technic. I subscribe wholeheartedly to the thesis that, leaving things to chance is the item which deprives patients of their lives.

Dr. Dunphy spoke of accelerating the descent of the tube. That certainly is an objective that we must strive to accomplish. It is an objective that we should learn to use with surgery; moreover I think the time will come when we may be able to empty the gut at operation with the help of an experienced intubator, work-

ing at the head of the table, such that the bowel may be as effectively decompressed as though the surgeon did an aseptic decompressive suction enterotomy.

Dr. Nowak spoke of back pain in strangulating obstructions. When traction occurs on the mesentery, at operation under local anesthesia, the pain is referred usually to the umbilicus. I suppose we would have to admit we do not know the mechanism of back pain in strangulating obstructions. We have all observed its occurrence, however, I would be more inclined to believe there is some irritation of the posterior parietal peritoneum. I am reminded that Dr. Nowak's patient had a leiomyoma of the gut in which this phenomenon was observed.

The whole picture of extrarenal uremia is a very interesting one, and we surgeons create the picture ourselves many times. Whereas the latitude of tolerance for abuse on the part of a young man to badly directed fluid therapy is almost without limit, the latitude of tolerance of the old man on this score is limited indeed. There is a syndrome of convulsions occurring in patients with mild hypochloremia in the upper age brackets; my patient, Thompson, referred to in this discussion had it. I believe we must set ourselves the task of meeting the electrolyte and water requirements of old patients, upon whom we do major surgery, right on the nose. We must learn to prevision exactly what those patients need and meet that requirement day by day. Long lag intervals during which the requirements have not been properly assessed and met, must not be allowed to intervene. The situation must be adjudicated many times a day, and in this assessment, use of the weighing scale is very helpful. I would not do without it.

The whole adynamic ileus problem, of course, is a very interesting one and a very difficult one; I suppose the most difficult of all the distentions which occur after operation. What brings them about? The patient had an indwelling tube and presumably the suction has been working. Why did the patient distend? Yes, we must get more satisfactory answers to such questions. Nor is the question of distinguishing a mechanical obstruction from an adynamic ileus after operation easy. The stethoscope should tell us. But it isn't that easy; you may have to listen long and return to the patient many, many times and listen again. You get what help you can from x-ray films. You might use pitressin; yet you worry about the suture lines and the possibility of disseminating limited peritoneal soiling, if it may be the source of trouble. You might use hypertonic saline solution intravenously, as suggested by Hughson and Scarff in 1924. One might use spinal anesthesia; yet we know that spinal anesthesia affords essentially only a preganglionic release of sympathetic influence. It does not contract the gut as effectively as does postganglionic removal of the sympathetic influence.

My associates and I have been toying with the idea, for a year or more now, of trying to inject the celiac and superior mesenteric ganglion to augment the contractibility of the atonic gut in adynamic ileus. Technical means may be devised to deposit some procaine, in patients exhibiting instances of adynamic ileus, in front of the aorta in the vicinity of the celiac and superior mesenteric sympathetic ganglia.

Yes, I have always been a keen and ardent supporter of the use of enterostomy, but I have lost patients through the employment of blind enterostomy, and I have come to learn how important it is to see what one is doing. I do not like to leave things to chance. Since 1942, I have been employing what I like to call aseptic decompressive suction enterotomy. The method is described in my book on bowel obstruction. The virtue of the method lies in the circumstance that the gut may be emptied at operation (and aseptically too) permitting visualization of the nature of obstruction. Yes, there are indications for the employment of blind enterostomy, but they are few indeed.

The Chairman spoke of spastic ileus. Yes, one must be alert to its occurrence. You will recall that I spoke of two patients in whom we had undertaken operation in the past two years for the relief of mechanical obstruction, in whom none was found at operation. They undoubtedly had spastic obstruction simulating closely all the signs of a mechanical obstruction. A little more persistence with suction applied to an indwelling tube undoubtedly would have resolved the situation satisfactorily. But how is one to know?

I have not observed the situation which the Chairman described as a complication of spastic ileus; however, I have seen a patient during the past 20 years, who was not operated upon because no obstruction could be demonstrated in the colon with a barium enema. Yet that patient died of colic distention and no obstructive lesion was found at autopsy. In other words, it is not safe to allow great distention to go unrelieved. The gut must be decompressed no matter what the cause of the distention is: this is true of mechanical, spastic as well as adynamic distention.

The discussion has been instructive to me. The progress from year to year may be very slow. I think as we look backward—and, mind you, it is easier to look a hundred years backward than it is to look one year forward, but, as we look backward, in spite of the circumstance that progress is slow, it is apparent we are inching forward with the bowel obstruction problem.

SURGICAL TREATMENT OF DUODENAL ULCER: A COMPARISON OF THE RESULTS OF TREATMENT WITH AND WITHOUT VAGOTOMY*

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When Dr. Dragstedt first cut the vagus nerves, he started a controversy, and everyone chose sides.

Many experienced gastric surgeons who, over the previous decades had developed a fine standardized technic for gastric resection, were loath to undertake a new operation. Some of these surgeons condemned vagotomy before they themselves had given it a fair trial. They warned of unknown dangers, physiologic and otherwise, which might attend the severing of these important nerves. Yet the mortality rate of gastric resection, for duodenal ulcer, ran from 2 to 3 per cent in the hands of experienced surgeons and many of the surviving patients experienced a morbidity which depended to a certain extent upon the type of operation performed, but which was constant. If the gastric resections were very radical, they were attended often by the problems of malnutrition, by the "dump" syndrome, and by the necessity of taking small, divided feedings. If the operations were not so radical, the gastric resections were followed by a high incidence of recurrent ulceration.

One of the reasons why certain thoughtful surgeons have rejected vagotomy is because they have taken pains to select from the groups of patients requiring surgical intervention a special group in which they have employed vagotomy. Among these surgeons is Mr. Rodney Maingot, of London, with whom I spent most of a long day discussing why we had come to opposite conclusions regarding the efficacy of gastrenterostomy and vagotomy in the treatment of duodenal ulcer. Finally it appeared that Mr. Maingot, a gastric surgeon of vast experience and outstanding skill, had selected for vagotomy those patients who, he said, had the "neurogenic" type of ulcer, in which the symptoms were greater than the physical findings at the time of operation. He hesitated to subject to a gastric resection a patient who had little or no evidence of ulcer, and likewise he hated to subject a private patient who had a large penetrating crater to an operation such as vagotomy and gastroenterostomy which was not yet tried and proved, when he knew that gastric resection gave an excellent chance of curing the ulcer.

I think everyone would agree that when you cannot demonstrate the characteristic intraabdominal pathology, whether it be in the treatment of gallbladder disease, or whether it be in the treatment of duodenal ulcer, the results which will follow will be unsatisfactory, mainly because in these cases are included all of the patients in whom the diagnosis was wrong. Certainly vagotomy is no treatment for functional gastrointestinal symptoms. Vagotomy does not help chronic gastritis. Vagotomy is a treatment for duodenal ulcer.

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Some of those who have been critical of vagotomy are surgeons who have done only a few of these operations. It is possible that these surgeons have not as yet developed a technic that assures a denervation complete enough to give good protection against the persistence of ulcer. Groups of fifty or one hundred cases, in which the patients have been operated on by several different surgeons, no one of whom has done many of the operations, do not afford a good evaluation of the full potentialities of vagotomy. The results may be as unsatisfactory as the results of gastric resection would be if one counted only the first five or ten patients upon whom any one surgeon did this operation.

One of the most careful students of the physiologic and pathologic consequences of vagotomy you heard yesterday, Dr. Francis D. Moore, of Boston, and I believe that a very worthy purpose has been accomplished by his carefully controlled studies of gastric physiology. Fundamentally, however, these problems are research and not clinical, because the vast majority of the operations in this group have been performed by transthoracic resection of the nerves, totally disregarding gastric, duodenal or associated intraabdominal pathology and without making any attempt to drain the denervated stomach. I believe the trend is well established throughout the country that transthoracic vagotomy is unsatisfactory, because it does not allow full evaluation of the intraabdominal condition and results in a high incidence of gastric retention with inevitable side effects.

Among those who have made outstanding contributions to our knowledge of gastric physiology is Dr. Walters who has studied intensively and carefully a large series of patients subjected to vagotomy. Unfortunately, however, Dr. Walters has not said what his indications for the operation were, whether his series is a consecutive series of cases, or whether he elected to treat certain patients by resection and others by vagotomy. In his reported series, moreover, are a number of gastric ulcers unseparated from the duodenal ulcers in the tabulation of results. Some of his vagotomies were done as vagotomies alone without a coincidental drainage operation and some of these were transthoracic. In his most recent publication[1] he reports 68 cases in which the patients were subjected to vagotomy, and in some of which gastroenterostomy also was done. Among these he lists 11 failures, but reading carefully through the article one can find that in at least 8 of these 11 failures the operations were done for gastric ulcers, were done as vagotomies alone, without drainage of the denervated stomach, or were done on patients who had had previous enteroenterostomies below a gastroenterostomy, a situation which with or without vagotomy is always attended by a high incidence of recurrent ulceration.

If one analyzes Dr. Walters' figures, on the basis of the operations which were done for duodenal ulcer and in which gastroenterostomy was done with the vagotomy—there appear to be only three patients who have not done well.

I call this to your attention because one must read all of these reports carefully to make certain that the authors are not including gastric ulcers and other lesions which are not amenable to treatment by vagotomy.

In general, the surgeons who have done gastroenterostomies or pyloroplasties along with vagotomies have been well satisfied with the results. These surgeons

realized that the denervated stomach did not drain satisfactorily for the first three or four months, and that the morbidity of the operation was too high unless it was accompanied by some type of a shunt. They also recognized that gastroenterostomy alone did something to correct the tendency to develop ulcer, that vagotomy alone did something to correct the tendency to develop ulcer, and that the combination of these two operations gave a synergistic effect in which the results were much better than those obtained with either method alone. For these reasons the results in a large number of patients treated by vagotomy combined with either pyloroplasties or gastroenterostomy have been highly satisfactory.

Perhaps the greatest danger of the operation of vagotomy and gastroenterostomy is its simplicity. Prior to the development of this procedure, surgeons who were not specially trained in gastric surgery often hesitated to undertake the risk of perhaps an 8 or even a 10 per cent mortality which a gastric resection for duodenal ulcer carried in their hands. They knew that gastroenterostomy alone did not give them very good results and, consequently, they were likely to refer these patients to others for treatment.

Because the operation of gastroenterostomy and vagotomy is simple and relatively safe, it will be a temptation for surgeons of limited experience in gastric surgery to treat patients by this means. If the indications for these operations are not sound, nothing but discredit will reflect upon the operation. We believe, therefore, that in spite of the simplicity of the operation, it should be reserved for those patients who have complicated duodenal ulcers. We should not extend the use of vagotomy and gastroenterostomy to patients with symptoms of ulcer, without demonstrable ulcers, and we should continue to limit it to the same group of patients in whom we formerly were willing to accept the greater risk of a gastric resection.

During the past three and one-half years the late Dr. T. E. Jones and I have treated by vagotomy something over four hundred patients, but many of these have not been followed long enough to make an analysis of the results at all significant. Almost all of these patients have had either a pyloroplasty or a gastroenterostomy done in association with the vagotomy. These represent less than 15 per cent of all patients with duodenal ulcer observed on the gastrointestinal service during this time.

We did four or five transthoracic vagotomies in the beginning, encountered gastric retention and recognized that it was not a sound method of approach. We observed the fact that jejunal ulcers healed promptly following vagotomy, and we could not see why there would be much risk of developing marginal ulcers after vagotomy and gastroenterostomy when a marginal ulcer was cured by the operation of vagotomy; therefore, from the beginning we did gastroenterostomies along with the vagotomies.

The first hundred patients have now been followed for a considerable length of time, and we have evaluated their progress carefully. The gastroenterologists of the medical division have made this evaluation under Dr. E. N. Collins' direction. and I should like to report to you the results obtained in the first 100 cases.

One patient died in the hospital following operation.

One patient died later of totally unrelated causes, about six months after the operation.

Three patients were lost in follow-up within the first year.

That leaves ninety-five patients out of the first hundred who have now been followed for from one to three years. The average duration of the follow-up in these cases is now two years. These patients, after the first six weeks, are given no diet, no restrictions are placed on smoking or drinking, and they are given no medicine. They are told that they are well; that they no longer have an ulcer diathesis.

The average age of these patients was forty-seven years, and duration of ulcer symptoms 12.6 years.

Bilateral vagotomy plus gastroenterostomy was done in 73, and pyloroplasty in 27 of this group. There has been little difference in the end results in the two groups of cases.

Since the pyloric end of the stomach is held up by the gastrohepatic ligament, the stomach distends after the vagotomy, and hangs in the pelvis even when the pylorus is patent. Drainage does not go uphill when the patient is standing or sitting so the patients have a tendency during the day to have retention and belching. Gastroenterostomy is like putting a spigot at the bottom of a barrel; it drains out the contents, and these patients have none of the symptoms of retention.

Prior to operation, 7 per cent of these patients had pyloric obstruction with from 30 to 100 per cent retention at the end of six hours. Massive hemorrhage, requiring hospitalization or transfusions, had occurred in 21 of the patients, perforations had occurred in 10, and there was a history of multiple perforations in two. This group of patients had complicated ulcers, not the ordinary ulcer in which the patient says he has a pain in his stomach, and the x-ray shows a duodenal deformity. These were intractable ulcers which could not be controlled medically.

The patients were asked the ensuing questions two years or more after operation:

"Have you vomited? How often?"

"*Yes*"—15 patients. That meant that 15 patients admitted vomiting one or more times from the time the patient left the hospital until the present.

Two vomited repeatedly.

Three had transitory vomiting which subsided completely in a few weeks.

Ten had mild vomiting and that is defined as one to six times a year. That is not very much vomiting.

"*No*"—80 patients.

"Have you had diarrhea? If so, how many stools a day?"

Thirty-five said they had had transitory or occasional diarrhea. I think anyone has occasional diarrhea. We may have a day or two in which we have four or five stools. That was classified as occasional. Six were classified as mild, and by that I mean the stools were loose or the patient may have had three or four a day. Two said "severe", which was six stools a day, but these patients were not incapacitated by the diarrhea; they were back at work.

There were sixty patients who had no diarrhea, and some complained of constipation.

(If you analyze a group of gastric resections, you will find much the same in respect to diarrhea.)

"How much weight have you gained?"

Fifty-eight patients gained weight—sixteen patients, no change; eight patients lost weight, but four were on a weight-reduction diet, and the other four lost six pounds. Thirteen patients did not state.

"Have you had any recurrence of your ulcer-like pain (pain in the upper abdomen two or three hours after meals and relieved by food)?"

A patient who has had a peptic ulcer for two years knows what you mean when you ask that.

Three patients in the group had had such pain, and 92 had no recurrence of their ulcer-like pain.

We then classified these results as excellent, satisfactory, improved, or failure.

In the excellent group we included only patients who were 100 per cent normal from the gastrointestinal standpoint. There wasn't a mark on their questionnaires except comments such as "I never felt better in my life".

"Satisfactory" was no ulcer distress; gastrointestinal symptoms so mild they do not interfere with work; no special diet or medication used. If a patient said he occasionally belched after meals, that would not be an "excellent" but a "satisfactory" result, because he had some gastrointestinal symptoms which he mentioned in the questionnaire.

"Improved" means no ulcer distress. I wish to emphasize that in the improved group the patients have no ulcer distress, their gastrointestinal symptoms are less severe than before operation, but they are employing diet or medication to control the symptoms. The patient with duodenal ulcer tends also to have disturbances of colon function which in most cases accounted for the residual abdominal symptoms. The distress of which the patients complained was lower abdominal distress, for which, however, they were taking some form of dietary or medical management.

"Failure" is any patient who has a persistence of ulcer-like distress or who developed side effects such as diarrhea severe enough to constitute a disability or prevent him from carrying out normal activities.

When these are broken down, we find 62 are excellent and 20 satisfactory (that means excellent plus satisfactory patients with no appreciable symptoms whatsoever constitute 86 per cent); 10 per cent are improved, and again I emphasize they have no ulcer symptoms but require some form of dietary or medical management to control such symptoms as irritable colon; and, finally, a group of three patients classified as failures, in each instance because they had a persistence of ulcer-like distress.

Next, we have analyzed a series of 87 patients treated by vagotomy with gastroenterostomy or pyloroplasty and have compared the results obtained with those which were obtained in a group of 87 patients treated by gastric resection or gastroenterostomy in the years immediately preceding the advent of vagotomy. It

is not fair to compare the vagotomy series with a series of cases in which the patients were treated with gastric resections alone. Most surgeons do eight or nine out of ten operations for ulcer as gastric resection, and the other tenth they do as gastroenterostomies. You cannot use the gastric resections alone as a comparison because the reason they did gastroenterostomy in the first place was that the patient was a poor surgical risk; so you must compare the way ulcer was treated before vagotomy was used—with the way it was treated afterward.

We were doing gastric resections on about four-fifths of our ulcer patients before vagotomy was used, and gastroenterostomies on one-fifth. This is the way it worked out:

Approximately the same number of patients was followed, and the follow-up was the same. If the maximum period of follow-up in the vagotomy group was eighteen months, we then disregarded any recurrent ulceration or late symptoms which might have occurred in the gastric resections or gastroenterostomy group occurring after that period of follow-up. The two groups were comparable from the standpoint of the duration of the follow-up.

The average age was the same; the duration of the symptoms the same. Duodenal ulcer was found at operation in all cases; intractable epigastric pain was present in all; night pain was present in 19 of the vagotomy group, and in 20 of the gastric resection and gastroenterostomy group; 29 of the patients with vagotomy bled before operation, as compared with 29 of the gastric resection group; there were 8 perforations in the vagotomy group, and 6 in the gastric resection group; and 27 and 29 respectively had pyloric obstruction.

So you see the two groups of cases in regard to severity of the disease, were exactly the same, as nearly as we could judge.

A questionnaire previously had been sent out to the gastric resection patients and we sent the same questions to the ones with vagotomy. In the latter group 4 per cent had ulcer-like pain, as compared with 18 per cent of the gastric resection groups. The incidence of persistent symptoms of ulcer was four times as high in the gastric resection group and most of these patients were continuing to take some form of antacid or dietary management to control it. That, to me, is a very significant finding.

Our gastric resections were about average in scope. We removed two-thirds to three-fourths of the stomach. It wasn't an extremely radical gastric resection nor a pylorectomy. Most of the stomach was removed.

In the vagotomy group one jejunal ulcer developed over this period of three years, and in the gastroenterostomy group there were three, all recurring after gastric resections. Four hemorrhages occurred after resection or gastroenterostomy and there were no hemorrhages in the other group.

There have been, altogether in the entire four hundred-odd cases, only two patients who have had any gastrointestinal bleeding since their vagotomies were performed, in spite of the fact that over 20 per cent of them had bled before. This figure is really amazing because none of these patients, not even the two that bled, had severe hemorrhages; neither of them required transfusions. One of them

occurred within a few weeks after operation and no ulcer was demonstrable in that case, nor in the other case was any ulcer demonstrable by x-ray. One patient had no symptoms at all—a few tarry stools and that was all. Vagotomy and gastroenterostomy have been most satisfactory in the control of the objective manifestations of the disease.

Now, let us compare the results in the two groups of cases.

Following gastric resections 19 per cent of the results were classified as failures, usually because of persistence of ulcer-like distress, as compared with 6 per cent failures following vagotomy and gastroenterostomy or pyloroplasty.

Gastroenterostomy alone showed exactly what you would expect, a 50 per cent incidence of failure in that group.

Now, I would like very briefly to run over with you the technic that we have been using for these operations. The incision is midline. We do not isolate the esophagus. We feel that that is dangerous. You are not attempting to denervate the esophagus; you don't want to. You might develop cardiospasm if you did. What you are trying to do is get the nerves which go to the stomach itself. It is not necessary to reflect the left lobe of the liver medially. All that is necessary is to pull down strongly on the stomach, palpate the vagus trunks through the peritoneum at the level of the diaphragm, pick a little hole in the peritoneum with a nerve hook, run a little nerve stripper up into the mediastinum to sever any branching filament of the nerve, and resect about an inch or an inch and a half of the nerve—you don't need to ligate the end. A search is then made for additional filaments which may be present—sometimes you find as many as six or seven. It is not necessary to close the peritoneum afterwards.

The procedure of vagotomy itself, after exposure is obtained, requires not more than about five minutes. The operation is very simple and it is based on the major premise that what you are trying to do is not to denervate the esophagus, but the stomach, therefore, if you pull down on the stomach, it puts the nerves under tension, because the vagus nerves are not elastic. The blood vessels, the musculature of the gastrointestinal tract and the peritoneum are elastic. The vagus nerve is not. It stands out like a violin string and can be palpated and picked up.

With gastroenterostomies it is absolutely unnecessary to have tubes in the stomach for a long period of time because if a gastroenterostomy is done, and if this gastroenterostomy is a properly functioning one, the stomach does not dilate, and it drains rapidly. The tube is taken out the morning after operation. Aspirations are done on the following two nights. If the aspirations show nothing over 100 or 200 c.c., no further aspirations are done, and that is usually the case.

A retention diet is started the same day, a full soft diet on the fourth day, and the majority of the patients are discharged on the sixth postoperative day on a simple diet. After the sixth week there are no dietary restrictions of any kind.

We have done 257 consecutive vagotomies with gastroenterostomy or pyloroplasty, without a death. I am not aware of any comparable series of gastric resections for duodenal ulcer that has been reported without a death. This operation, therefore, is relatively safe. I believe if you can do 257 consecutive vagotomies with-

out a death—and our over-all mortality is well under 1 per cent—that the mortality rate should be about 0.5 per cent.

In this consecutive series we have not had to reoperate upon a single patient for obstruction of the stoma, or for obstruction of any kind. All of these patients, as I have said, have had good function of their gastroenterostomy stomas.

In conclusion, I wish to emphasize that in our hands gastric resection for duodenal ulcer has entailed a higher mortality; a higher morbidity; and, third, a lower curability of ulcers than follows vagotomy and gastroenterostomy. We attribute these results to three factors: (1) We have employed a posterior gastroenterostomy situated at the most dependent aspect of the greater curvature to drain the denervated stomach. (2) We have employed vagotomy and gastroenterostomy in all cases of duodenal ulcer in which elective surgery is necessary and hence have not selected a special group of cases for this operation, and (3) Vagotomy performed by the technic described is complete enough to give a high degree of protection against recurrence of ulcer.

REFERENCE

1. Walters, W., and Fahey, M. M.: "Vagotomy: immediate results in 28 cases and later results in 68 cases." Proc. Staff Meet. Mayo Clin. **24**:501 (Sept. 28), 1949.

Discussion

Dr. James T. Nix (New Orleans, La.):—Dr. Crile is to be congratulated upon his able presentation of this significant contribution to a most controversial subject. My remarks will consider some of the reasons for the healthy difference of opinion among surgeons concerning the role of vagotomy in the management of complicated duodenal ulcer.

There are many question marks in gastric physiology. The Edkin's hypothesis conveniently divides acid secretion into a cephalic, a gastric and an intestinal phase and there agreement ends. The interrelationship of these individual phases is shown by recent evidence suggesting the mediation of the cephalic phase through the pylorus. Vagotomy attacks the cephalic phase, resection the gastric phase and enterogastrone the intestinal phase. Internists and surgeons center therapy about the phases of gastric secretion for lack of a direct method of blocking the formation of hydrochloric acid by the parietal cell.

How widespread are the effects of supdiaphragmatic vagotomy? How lasting? The effect of vagal section on the intestines and pancreas remains to be clarified. Wollager studied fecal fat and nitrogen in three vagotomy subjects and found no impairment of digestion or absorption. However, additional observations on intestinal and pancreatic function are needed. Furthermore, it is not known what complications might follow section of this parasympathetic trunk in patients with essential hypertension. Likewise, the permanence of changes in acid secretion and motility may well be questioned. In the most recent report of Dr. Walters, gastric motility returned in 80 per cent of patients within eighteen months after vagatomy.

How much reliance can be placed on the insulin test as a measure of the completeness of vagotomy? The vagus has many actions on the stomach (acid secretion,

motility, mucus production). As the insulin test measures only acid secretion, it can hardly be accepted as an index of total vagus function.

Clinical studies show many variables in the selection of patients and procedures, and in the methods of measuring results. As gastroenterostomy alone is still an acceptable operation for older patients with pyloric obstruction and low gastric acidity, any improvement following a combination of vagotomy and gastroenterostomy may well be credited to either procedure. This comment applies to the twenty-four operations for pyloric obstruction in the series of Dr. Crile.

Follow-up studies on this and similar surveys will determine the duration of the suppression of acid secretion and define the role of vagotomy in ulcer therapy. Passing years will write their judgment.

Dr. M. E. Steinberg (Portland, Ore.):—It is my opinion that generally, the indirect gastric operations with or without vagotomy are not as promising as the well planned gastrectomy. The choice of the type of gastric operation for simple ulcer is determined by mortality rate, incidence of morbidity in the immediate postoperative period, the assurance that the original ulcer will be permanently cured and that the incidence of jejunal ulcers will not be prohibitive. Though much attention has been given to the postgastrectomy syndrome, the side-effects which follow pyloroplasty, gastroduodenostomy, and gastroenterostomy have received little consideration. Alteration of function results not only from loss of tissue and organs but also from the new continuity established between the stomach and intestine.

Some crippling side-effects and the frequent occurrence of jejunal ulcer are unmistakably the result of choosing the wrong type of gastric operation. Physiological dislocations, however, take place after every gastric operation. The freedom from annoying symptoms and disabilities even after the proved technic, largely depends on the patient's ability to compensate for radical changes of function brought about by the operation.

My introduction to the indirect operations of pyloroplasty and gastrojejunostomy came through observation of 97 patients who each had from 1 to 8 antecedent operations resulting in failure. In seven of these patients the original unsuccessful operation had been performed by me. Collectively these 97 patients had had 146 unsuccessful antecedent gastric operations.

My last gastroenterostomy was performed in 1925. Since then 558 patients have undergone gastrectomy for benign ulcer. This number includes the 97 patients with 146 antecedent gastric procedures. The mortality is 1.4 per cent in 558 consecutive unselected gastrectomies beginning in 1924. Four hundred and fifty-seven patients were treated by the subtotal conventional retrocolic gastrectomy with a short proximal jejunal loop placed at the lesser curvature of the gastric remnant. After this type of gastrectomy only two of these 457 patients returned with jejunal ulcers. These had revision of gastrectomy with good results. Two other patients from the same group were operated elsewhere, presumably because of jejunal ulcer. It is most unlikely that this number represents the true incidence of jejunal ulcers after subtotal resection by the Hofmeister-Finsterer technic. Fourteen patients have had subtotal gastrectomies with the exclusion of the ulcer by leaving a part of

the pyloric antrum with the mucosa not ablated. Four of these 14 patients which were originally operated by me returned with jejunal ulcers. They were reoperated successfully. Nine patients who had developed jejunal ulcers following gastroenterostomy performed elsewhere were subjected to subtotal gastrectomy with the use of a proximal Y-anastomosis and with a long proximal jejunal loop, a modification of the original end Y-anastomosis of Roux. Two of these patients returned with jejunal ulcer. They were reoperated with good results. It is obvious that a subtotal retrocolic resection with a short proximal jejunal loop transcends other gastrectomies in the promise of cure for the original ulcer and the prevention of postoperative jejunal ulcer. I have had no hesitation in assuring the patient that his gastrectomy will probably not result in jejunal ulcer. However, I experience no such comforting poise when I take up the side-effects of this operation with the patient. The manifestations of the postgastrectomy syndrome are numerous and protean in nature. Some of the intractable and annoying symptoms result from excessive reflux of irritating pancreatic juice and bile into the gastric pouch producing gastritis with subsequent nausea, anorexia and vomiting. Since 1932 I have been experimenting with a modified gastrectomy operation which creates a larger jejunal pouch with formation of a valve at the gastrojejunal junction. It aims to prevent reflux of enteric contents into the gastric pouch and to add storage capacity to delay transfer of foodstuffs. This operation has now been used in 78 patients with ulcer, in 6 patients with cancer, and in 2 total gastrectomies. It has also been employed in 5 patients who had developed severe intractable symptoms after the conventional gastrectomy. All of the 5 patients reoperated by the new method gained weight and made an excellent recovery. I gain the impression that such minor complaints as intolerance to milk and sweets and the inability to take large meals do not differ much from those encountered after the established operation. Thus far, however, none of the 86 patients with the new gastrectomy operation have displayed any semblance of the grave symptoms and disabilities characteristic of the so-called dumping syndrome or "jejunal shock". The most significant results were obtained in those 5 patients who developed severe and intractable symptoms characteristic of "jejunal shock" and severe gastritis after the established operation and who completely recovered after being reoperated by the new technic. I am now using this new pantaloon operation routinely for ulcer and cancer. Apparent normal health and freedom from annoying symptoms after gastrectomy does not imply full restoration or compensation for altered function.

My attitude to the various surgical procedures used in the treatment of simple ulcers has crystallized from personal experience, from personal laboratory experimentation and from interpretation of physiological studies and clinical reports from numerous other sources.

The healing of duodenal ulcer cannot be assured by gastroduodenostomy and by pyloroplasty for the obvious reason that these operations do not shunt the acid gastric contents away from the duodenum. Contrary to prevailing opinion, the reflux of alkaline duodenal content into the stomach is undesirable because it is harmful to the gastric mucosa. No sustained reduction in acidity takes place after

the complete reflux of the alkaline enteric content into the stomach by the Schmilinsky operation. On the contrary the Schmilinsky operation results in jejunal ulcers with regularity in the experimental animal and in the patient. Duodenal ulcers cannot always be removed because they are multiple and because they are frequently situated on the posterior wall. A successful pyloroplasty or gastroduodenostomy is not possible in the presence of distortion and cartilagenous inflammatory tumor formation encroaching on the vital structures of the duodenohepatic mesentery. Such objections also apply to the various modifications of the Billroth I resection some of which are presently being revived with enthusiasm.

Neither does gastroenterostomy assure the healing of duodenal ulcer. Duodenal ulcer may bleed or perforate. The healed duodenal ulcer frequently recurs. Jejunal ulcers cannot be prevented with the same degree after gastroenterostomy as after a well planned gastrectomy. Complications from stasis are more frequent after gastroenterostomy than after gastrectomy in the immediate postoperative period. Side-effects following the indirect gastric operations and all the modifications of the Billroth I gastrectomy result in minor or graver symptoms not unlike those which take place after the Billroth II gastrectomy. Granting that a concomitant vagus resection does exert a definite improvement in the healing and in the prevention of recurrence of the original ulcer and in the prevention of the jejunal ulcer, it cannot preclude the side-effects of these operations. There is some evidence that vagotomy is followed by side-effects of its own making.

Gastrectomy has been most successful in my experience in curing the original ulcer and in preventing a jejunal ulcer. Side-effects after gastrectomy resulting in minor symptoms are not infrequent. Intractable and annoying symptoms result in a small percentage of patients. The new pantaloons operation gives promise of either abolishing or ameliorating some of the symptoms peculiar to the postgastrectomy syndrome.

It is apparent that my surgical approach to the ulcer problem is different from that of Dr. Crile's. Nevertheless, I consider Dr. Crile's experience with the vagotomy operation as a major contribution to our concepts and knowledge to the problem of surgical therapy for peptic ulceration.

Dr. Carl Bearse (Boston, Mass.):—Up to the revival of interest in vagotomy —and some of our purists in Boston think that the term really should be "vagus resection"—gastric resection had become the preferred operation for intractable duodenal ulcer. I use the word "revival" advisedly, for while the current wave of enthusiasm for vagotomy dates back to the operative work of Dragstedt, beginning in 1943, his experimental work going back to 1924, observations on the effect of section of the vagus nerve had been made as far back as 1814, by Brodie, an English surgeon, and this work had been confirmed by Wood, in 1870, and by Pavlov in 1894.

As pointed out to us by Dr. Crile, gastric resection is still an operation of magnitude, and is not altogether free of serious complications. It is therefore not surprising that vagotomy excited the interest of the medical profession, not alone because vagotomy is a less complicated procedure than resection, but also because it

appeared to have a more physiologic basis. Those of you who have heard Dr. Crile describe the operation of vagotomy—well, it seemed pretty simple, and I can testify to the fact that I had occasion to view two of these operations at the Cleveland Clinic a couple of years ago, and they certainly made this operation look very easy.

In appraising the merits of vagotomy, a comparison of the end results with those of gastric resection is indeed instructive, and we should be grateful to Dr. Crile for coming to Boston and for giving us such a most informative presentation. Granted, however, that the preliminary reports on vagotomy are encouraging, it must be conceded that it is still too early to evaluate the effect of the operation on the prevention of recurrences.

Even a follow-up of six years, back to Dragstedt's early operations, is hardly long enough to give us a true picture of its long range value. As we all know, patients have remissions for many years following all sorts of therapy. Many of you have undoubtedly had patients who have had remissions for many years following medical treatment. Psychiatrists tell us that they have seen benefit for many years just following their specialized form of therapy, and certainly we have seen remissions for many years following gastroenterostomy alone—nothing else added to it.

I have several patients who had a gastroenterostomy done twenty and more years ago, and have been free of symptoms. A conservative approach, therefore, it seems to me, should be taken regarding vagotomy, whether it is with or without gastroenterostomy or pyloroplasty. The reason that gastroenterostomy fell into disrepute was the high incidence of postoperative complications, particularly marginal ulcer, and gastrojejunocolic fistula.

Enough time must be allowed to elapse to know whether vagotomy may not also be followed by recurrence, hemorrhage, perforation; whether gastric acidity may not return and be followed by marginal ulcer and gastrojejunocolic fistula when gastroenterostomy has been added.

Unanswered also are the long range effects on the other organs supplied by the vagus nerves such as possible disturbances in intestinal function; the effect of partial denervation of the pancreas and duodenum; and what has been termed as the "loss of receptive relaxation" of the duodenum; not to be overlooked is the remarkable ability of the autonomic nerves to regenerate.

Now, I do not mean to imply that vagotomy should not have been done. It certainly deserved a trial, but we should not be too hasty in reaching a decision as to its value. It took a long time before gastroenterostomy was given up for resection —about twenty years. The suggestion made, I believe it was by Eustermann, of the Mayo Clinic, that possibly a five-year moratorium be declared on vagotomy, deserves our consideration.

In January, 1948, Keith Grimson, of Duke, reported that more than five thousand vagotomy operations had already been done by various surgeons. This, it seems to me, is a large enough series to give us, in due course, a fairly accurate idea as to the merits of vagotomy and its place in the treatment of duodenal ulcer.

We shall look forward with much interest to further reports by Dr. Crile and others doing pioneer work in this field. Of course, the outlook for ulcer sufferers

would be much brighter if simpler means than operation were devised to overcome ulcer diathesis, but until a more adequate operation than gastric resection is available, it does seem, to me at least, that the problem of ulcer must be faced in the light of our present knowledge.

Dr. John M. T. Finney, Jr. (Baltimore, Md.):—I had not expected to be called on to continue the discussion. It came as a total surprise; however, I was very much interested in Dr. Crile's figures, and I should like, possibly for congenital reasons, to call attention to the fact that in the use of the vagotomy operation, there has been a reincarnation of the operation of pyloroplasty, that had fallen out quite a bit. I have not recently tried to collect statistics on our operations, but we have quite a group now which fall into the class which Dr. Crile speaks of as vagotomy with pyloroplasty.

The type of pyloroplasty we do, of course, as you might expect, is the rather wide, Finney type of pyloroplasty, not the Heineke-Mikulicz, or some of the lesser ones, and I believe that it has a very definite place now in connection with vagotomy. I believe that the wide stoma which one gets thereby does away with what has been spoken of by Dr. Crile just now as the tendency for food to collect for the first few weeks. After all, if one does the wide pyloroplasty type, the ultimate pylorus is placed down about the inferior angle of the reflexion of the duodenum, the inferior curve, so that it is in fact practically as dependent as when one does a posterior gastroenterostomy on the greater curvature.

I think we should all be conservative in our approach to this problem. Personally I believe that one cannot confine all stomach operations to one type of operation. Dr. Crile urges that we do that so that our figures will be consistent; however, I still cannot but feel that one should try to fit the operation to the patient rather than the patient to the operation in most instances, and, in doing that, I think there will be a necessity for using more than one type of operation, be it vagotomy, resection, pyloroplasty, gastroenterostomy, or what not.

Dr. George Crile, Jr. (Cleveland, Ohio):—In the first place, I should like to thank the discussers for their remarks.

I am in perfect agreement with Dr. Finney that you should select the type of operation for duodenal ulcer, but, since I am convinced that the pathologic physiology which produces this disease is mediated through the vagus nerves, I believe that vagotomy should be an integral part of the operation in all instances, whether one elects to do a gastric resection, a gastroesterostomy, or a pyloroplasty. I do not believe that there is any place for vagotomy alone, because of the high and often totally unexpected morbidity which occurs in patients who have not necessarily had any pyloric obstruction prior to the operation.

Dr. Steinberg must have misunderstood our results in respect to vomiting. There were two patients out of a hundred—2 per cent—who have vomited repeatedly since operation. Fifteen vomited from two to six times, that is, at the most, once every two months during the first year, and in analyzing a group of gastric resections by the same method, we found an equal incidence of vomiting from the

time the patient leaves the hospital on through, so there have been two, not fifteen, repeated vomiters.

Dr. Nix spoke of the motility of the stomach and acidity of the stomach after vagotomy, and interpreted the return of acidity and motility as signs of possible vagus regeneration. I do not believe that it should be so interpreted because we know that anywhere in the autonomic nervous system, whether it be a blood vessel or a viscus, the immediate effect of denervating an organ and isolating it from the central nervous system is to produce atony and lack of function. Very quickly thereafter the autonomous mechanisms in the organ begin to function. Tone and function are regained; however, there is no indication that the psychic stimuli, which is the pathologic physiology of ulcer, do return. Dragstedt has been unable to produce any return of psychic stimulation after section of vagi in dogs even when he anastomoses them, and the Hartzell experiment was not conducted to study the effects of higher centers or gastric secretion. It is merely normal acidity which returns. No one minds if normal acidity comes back; it is abnormal acidity one wishes to prevent.

Dr. Bearse's conservative stand is well taken. The final answer lies ahead, but nevertheless, the analysis of our results prior to the advent of vagotomy shows half of the recurrences took place within the first year. Only one patient in our entire experience with vagotomy has had a recurrence after six months. Any other recurrences have been failures right from the first few months.

If a five year moratorium were made on vagotomy, I think it would be a most unfortunate thing. The mortality rate of gastric resection in our hands, and I am quite sure throughout the country, is three times as high as it is for gastroenterostomy and vagotomy. Death is irreversible. During that five years many patients would have died unnecessarily. Vagotomy is not irreversible, in that medical treatment can be given if any ultimate and hitherto totally unindicated disturbances of gastrointestinal function take place.

THE ABDOMINAL INCISION*

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Surgery has passed from its anatomical infancy, through bacteriological and pathological childhood, to its present physiological teen-age. Operation today is like the meat in a sandwich; preoperative and postoperative management are its two pieces of bread. Surgery is continually pushing ahead to new goals. Consequently, the surgeon today who contemplates the making of an abdominal incision is faced with the whole problem of wound healing and all that makes an operation safer for the patient.

The object of an abdominal incision is to obtain adequate exposure with minimal damage to the abdominal wall. Vertical incisions are easily made and easily closed. The disadvantages to these types are: (1) difficulty in closing (when there is increased tension), (2) cutting of nerves and fascial fibres at right angles to their natural course. Transverse incisions are: (1) less destructive to tissues, (2) easier to close, (3) more comfortable for patients and (4) stand strain of coughing, vomiting and distention better. The disadvantages to these incisions are[1]: (1) possible inadequate exposure, and (2) they take longer to close. Oblique incisions are a compromise. A combination of incision types is possible.

The evaluation of surgical risk is the prime consideration. For this a complete history and mental and physical examination is required. Special problems may present themselves; such as, obesity, cardiorenal disease, diabetes and infancy and old age. In other words, we must treat the whole patient, not the surgical ailment alone.

In the wards of the Boston City Hospital we have sick people with almost every conceivable deficiency; mental, physical, financial, nutritional, vitamin, fluid, blood, cardiac, renal and what have you. All the needs of these patients must be adequately met; the spiritual and mental as well as the physical. Harmful effects of nutritional deficiencies of the patient are now being recognized and combatted. Yet, the study of nutrition as applied to surgery is still in its infancy.

Protein has an important role in nutrition. There are two kinds of protein: (1) tissue protein, cell protoplasm and nucleus, (2) plasma protein, globulin, albumin, and fibrinogen.

In process of digestion protein is broken down into amino acids and absorbed from the small intestine, through the liver to general circulation[2]. Amino acids are rebuilt as follows: (1) to replace tissue and (2) to build blood proteins, namely hemoglobin, globulin, fibrinogen and albumin. The end product of amino acid catabolism appears in urine as urea for the most part; that is, as nitrogenous waste.

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Protein deficiency is caused by inadequate intake, impaired absorption, hemorrhage, liver damage or high B.M.R. from fever, sepsis or trauma.

Carbohydrates and fat are protein sparers, i.e., they are used instead of body tissue protein to furnish energy. Also we must remember that food ordered for the patient but not eaten is of no value to the patient.

Protein deficiency causes slow wound healing, lessened resistance to infection, disturbed water balance; i.e., blood volume is decreased and nutritional edema ensues. It also causes weakness, anorexia, loss of weight, anemia, depression, etc. Protein is about 16 per cent nitrogen. The body utilizes its own tissues when a negative nitrogen balance exists. Therefore, more protein is needed in abnormal conditions. Many diseases seem to arise in a nutritional deficiency state. Parenteral alimentation is necessary when the patient cannot get a sufficient amount orally or cannot assimilate properly. Fortunately, amino acids can be given intravenously.

The use of protein hydrolysates is currently popular and helpful for supplying parenteral food. But these are no good for shock of hemorrhage. Serum protein determinations are a guide to protein needs. Protein is obtained quickly by whole blood transfusion when anemia exists and by plasma when anemia is not a factor. Limiting water with hypoproteinemia decreases the edema. Increase in sodium concentration causes water to be eliminated. Decrease in osmotic tension allows retention of salt and water in interstitial spaces when hypoproteinemia exists. Low salt intake allows kidneys to excrete water and salt. Protein is the major contributor to osmotic pressure in the blood stream.

Loss of blood may present a serious problem. Blood volume, after hemorrhage, is restored promptly by fluid (water and salts) drawn from the extravascular spaces. The average adult has about 6,300 c.c. of blood. Fifty-four per cent is a protein and salt solution called plasma. Loss of 30 per cent rapidly is usually fatal if blood transfusion is not given to replace the deficit. Therefore long operations may necessitate multiple transfusions.

The causes of blood volume reduction are (1) hemorrhage, loss of whole blood, (2) anemia, reduction in red cells, (3) plasma loss, as in shock, (4) water loss. With hemorrhage, watery fluid is drawn into the circulation with resultant reduction in concentration of r.b.c. and protein both.

Blood loss may be acute or chronic. The first indication is to stop the bleeding. Whole blood transfusion is the best quick remedy for blood loss. There is no satisfactory blood substitute. Nutritional anemia is helped by whole blood transfusions plus protein and folic acid. The dangers of whole blood transfusions are: (1) reactions due to technical errors or incompatibilities, (2) transmission of hepatitis, syphilis and malaria. Plasma is the best substitute for whole blood.

Fluid Balance:—The object of fluid administration in patients with fluid and electrolyte imbalance is to restore the intracellular fluids to the normal pattern. Osmotic pressure is one of the fundamental forces underlying many physiological processes; e.g., the excretion of urine, interchange of fluids between interiors of blood cells and their surroundings, as well as the regulation of blood volume[2]. The

flow of fluids from tissues into the blood stream and vice-versa depends on osmotic and hydrostatic pressure. The semipermeability of the membrane is an essential factor in the development of osmotic pressure.

The other important factor in the interchange of fluid between the blood and the general body fluids is the hydrostatic pressure within the capillaries, i.e., blood pressure. When blood volume is increased or decreased both the osmotic and the hydrostatic pressures are altered. These changes bring the blood fluid automatically to its proper level. Edema is the term applied to an excessive accumulation of fluid in the tissue spaces due to a disturbance in the mechanism of fluid interchange.

Dehydration is caused by (1) inadequate intake of water, (2) excessive sweating, (3) abnormal loss through vomiting, diarrhea, or draining from indwelling gastric tube, and (4) reduction in electrolytes in body fluids.

The signs of dehydration are: (1) dry skin and tongue, (2) scanty urine output, (3) hemoconcentration, (4) protein concentration, (5) rise in blood r.p.n. and (6) thirst. Water does not become available for urine formation until wants of the body are met. Up to 4,000 to 7,000 c.c. of water may be needed in some cases for replacement before excretion of urine begins. The kidneys play an important part in conserving water and salt, excreting excesses of either or both.

Intravenous isotonic glucose solution is the best way to replace water if the oral route is not available. Giving of excessive water to patients with anuria due to renal damage may cause water intoxication. Anuria of renal insufficiency must be distinguished from anuria due to inadequate fluid intake. Dangers exist in fluid administration. Patients with low protein levels or cardiorenal disease may be lost by unwise administration of saline solution. The patient's tolerance must not be exceeded. Cardiacs should have what they lose put back, be it blood, water or protein—no more and no less. Dehydration delays wound healing. The appearance of the patient and response to treatment are the most valuable guides. An intake and output chart is a must for serious surgical cases. An adequate output of urine is the best guarantee of fluid balance. A minimum of 1,500 c.c. is needed to eliminate nitrogenous waste.

NaCl is the most important salt in the extracellular fluid. Salt deficit arises from sweating, indwelling gastric tube, ileostomy, vomiting and diarrhea. Salt deficiency causes weight loss, weakness, hypotension, anorexia, poor wound healing, protein breakdown, low blood chlorides, altered metabolism, rise in N.P.N., high blood urea, kidney damage and uremia.

The tests for salt deficit are: (1) the amount of NaCl in the urine (drops first when renal function is normal), and (2) blood chloride.

The treatment is water and salt. Water alone is not enough. Sodium ion, not the chloride, governs fluid and electrolyte balance. The dangers of excessive administration of parenteral saline are suppression of urine, acidosis, pulmonary edema and death. There is a wide margin of safety in the patient with good kidneys and satisfactory urinary output.

It is the sodium ion rather than the amount of fluid given which causes retention of body fluid. Edema cannot occur without retention of sodium as well as water. Pulmonary edema is serious. Edema, the accumulation of fluid in the interstitial spaces is due to: (1) excess salt administration, (2) damaged kidneys. Edema retards wound healing. Contraindications to excess saline administration are hypoproteinemia, potassium deficit, cardiorenal disease, edema, hemorrhage or shock, and simple water deficit.

Potassium as well as sodium metabolism is intimately connected with water balance and acid base equilibrium. Potassium deficit results from excessive administration of saline solution, vomiting of intestinal obstruction, diarrhea with dehydration. According to Randall and coworkers at the Columbia Presbyterian Medical Center, hypochloremic alkalosis is a potassium deficiency state[3]. With a flame photometer they checked the levels of potassium in their patients. Alkalosis causes more potassium to shift from the intracellular to the extracellular spaces. The symptoms of potassium deficit are muscle weakness, distention, anorexia, irregular pulse, and respiratory distress. The treatment of potassium deficit is to give potassium orally or intravenously.

The adrenal cortical hormone also has a profound influence on electrolyte and fluid in the body. It regulates potassium metabolism and distribution. The advantages and contraindications to early ambulation deserve mention.

Our knowledge of vitamins dates from about 1912. Vitamins are essential to cellular metabolism. Vitamins C and K are of established value to the surgical patient. Vitamin C is needed for wound healing[4]. One to four grams daily may be needed in a severe deficiency state. Vitamin K is needed in various types of hepatic disease. Folic acid helps correct nutritional deficiency anemia. One injection daily of thiamin riboflavin and niacin helps the seriously sick patient. New knowledge may reveal more surgical importance of vitamins than at present.

Wound healing is influenced by the age of the patient, nutritional states, anemia, infection, surgical technic, blood supply, trauma and foreign bodies, such as talc. Talcum powder used for gloves has been proven to cause foreign body reaction of a serious nature in the tissues[5]. Corn starch powder is a satisfactory substitute.

The problem of sutures is intimately connected with wound healing. It is the size of sutures not the kind that counts. Skin does not tolerate absorbable sutures well. Fat and muscle tolerate sutures poorly and add nothing to wound strength. Fascia gives wound strength, (connective tissue). Many fine interrupted sutures are preferred to heavy sutures. Sutures are affected adversely by trauma, tension, poor hemostasis and infection.

Everyone connected with a surgical operation must be bacteria conscious. Culturing knife blades used to make the skin incision in a series of my surgical cases proved to me the necessity of proper draping of the skin edges. Contact of gloves, instruments, gauze, suture material or retractors with the skin, allows bacteria to be transferred into the wound. Sterilization of the deep skin is not reliable[6]. Air bacteria are a constant hazard especially when upper respiratory infections are

prevalent. Ultraviolet light has been used to sterilize the air of operating rooms with considerable success[7]. This, however, is a hazard to the eyes and skin.

Using a glass shield and special draping proved to me that a mechanical barrier is a satisfactory answer to the problem of air bacteria. I placed culture plates inside the barrier during operation and control plates outside. The difference in the number of colonies was amazing. Except for difficulty in sterilizing and its being a fire hazard, cellophane sheeting would be a good mechanical barrier. With a proper mechanical barrier, coughing, sneezing and talking would be permissible as well as close observation of the operation by students. It seems absurd to me to use a mechanical barrier over the surgeon's nose and mouth, while leaving the patient's wound open as a repository for air bacteria.

Surgical infections are a test of the patient's resistance versus the virulence of bacteria. Clean wounds are rarely entirely free from bacteria. Wound infections result from dead tissue, poor blood supply, hematomata, contamination, sutures too tight or too heavy and malnutrition and anemia. Signs of wound infection are postoperative fever and local changes in the wound, such as tenderness and fluctuation. Bacteria of wound should be recognized and antibacterial agents proved effective against the bacteria in the wound should be used. Chemotherapy and antiseptics are an aid, not a substitute, for sound surgical treatment. Despite pitfalls and abuse, the antibiotics are a tremendous aid in the prevention and treatment of wound infection. Penicillin and streptomycin are of proved value. Newer antibacterial agents, such as bacitracin, aureomycin, chloromycetin and others are being developed.

During 1945 and 1946 at the Boston City Hospital we performed a total of 3,038 abdominal operations with 13 cases of wound disruption. Evisceration is caused by insecure closure, strain of coughing or the like, distention, faulty healing due to local or systemic complications such as lack of Vitamin C and infection and hypoproteinemia. Signs of evisceration are serosanguineous discharge, swelling under the skin, and painful incision. Hernia results when only the peritoneum and posterior sheath of the rectus give way. Evisceration exists when all layers of the wound under the skin separate. Operation with sutures of wire including all layers of the abdominal wall is our usual treatment of wound disruption.

All this has been presented to prove that the surgeon who is contemplating an abdominal incision has many more things to think about than the use of his scalpel.

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SURGICAL DISEASES OF THE PANCREAS*†

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There is no other organ in the abdominal cavity, that beset by disease, may manifest its symptoms in as protean a manner as does the pancreas. The explanation is apparent. The pancreas is a retroperitoneal abdominal organ traversing the upper abdomen, and it is in proximity to large vascular structures. There is rich enervation present and the ducts of the pancreas joining the common duct above a common sphincter form an interrelationship in the cause, as well as the effect of disease in both the liver and the pancreas. The contact with various parts of the gastrointestinal tract produces in pancreatic diseases significant clinical manifestations in that tract. In disease as well as in disorders of function, the pancreas produces far reaching metabolic disturbances which are difficult to interpret, and which constitute a challenge to the therapeutist. Thus it is apparent that the symptoms of pancreatic diseases exhibit themselves in variable ways.

For the surgeon the following classification modified from that of Cattell and Warren[1] of the Lahey Clinic is not all inclusive, but basic and practical.

TABLE I

INFLAMMATION

Acute

1. Acute pancreatic edema or acute interstitial pancreatitis
2. Acute hemorrhagic necrosis
3. Acute suppurative pancreatitis
4. Pseudocysts

Chronic

Chronic relapsing pancreatitis

a. Chronic fibrosing pancreatitis
b. Pancreatic lithiasis
c. Pancreatic calcification
d. Retention cysts
e. Pseudocysts

TABLE II

TRAUMA

1. Traumatic Pancreatitis
   a. Operative
   b. Nonoperative
2. Pseudocyst
3. Fistula
   a. Internal
   b. External
4. Chronic Relapsing Pancreatitis

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TABLE III

TUMORS

Benign
1. Islet cell adenoma
2. Proliferative cyst adenoma

Malignant
1. Islet cell adenocarcinoma
2. Cyst adenocarcinoma
3. Adenocarcinoma
4. Duct cell carcinoma
5. Squamous cell
6. Sarcoma
7. Lymphoma

TABLE IV

SURGICAL PANCREATIC DISEASES

Congenital
1. Ductal
2. Accessory or ectopic
3. Annular
4. Congenital cysts
   a. Hemangioma, Lindau's disease
   b. Familial cystic disease
5. Chronic familial cystic pancreatitis
6. Dermoid cysts
7. Teratoma

ANOMALIES

1. *Ducts:*—Anomalies of the ductal system are the commonest developmental deviations. Opie[2] found that the two ducts, those of Wirsung and Santorini, may join together—may remain separate, or one or the other may become obliterated. In the majority of cases they come from a main duct that joins the common duct and empties into the duodenum through the Sphincter of Oddi.

2. *Ectopic pancreas:*—By this term is meant an entirely different isolated mass of pancreatic tissue from the main gland. Heterotopic tissue has been found in this order of frequency—in the stomach, duodenum, jejunum, ileum, mesentery, biliary tract and spleen. These findings are usually operative or postmortem. They are small and in the gastrointestinal tract in the submucosa, they may mimic an ulcer, and should be removed if possible. If they obstruct, palliative obstruction surgery is indicated. They do not undergo malignancy.

3. *Annular pancreas:*—There are 52 cases reported in the literature by Lehman[4], Chapman[5] and Gross[6]. The condition is characterized by a ring of pancreatic tissue around the second portion of duodenum. Failure of the ventral portion of the pancreas to rotate with the duodenum, and its union with the dorsal portion around the duodenum causes this rare *anomaly*. The symptomatology may be that of acute obstruction in the new-born, chronic duodenal obstruction in the adult, or recurrent obstruction in the adult associated at times with jaundice due to pancreatitis.

Treatment of these types is surgical and may consist of: (1) Division of Annular Ring; (2) Gastroenterostomy; (3) Duodenojejunostomy.

Theoretically, short-circuiting is a safer operation. It precludes the danger of hemorrhage, fistula and necrosis, as well as insuring against obstruction persisting from incomplete division.

Congenital Cysts

1. Hemangiomatous cysts involving the pancreas, kidneys, mesentery, cerebellum and other parts, known as Lindau's Disease, a rare destructive disease, familial and prone to malignancy.

2. A form of familial cystic disease involving kidneys, liver, pancreas and ovaries in the female, rarely the omentum, mesentery and peritoneum. The usual predominating symptomatology is that of kidney insufficiency. There is no therapy.

3. Chronic Cystic Pancreatitis. This is a fatal familial disease. Cystic fibrosis of the pancreas occurs. The tissue is destroyed and replaced by cysts of varying size and by fibrous tissue. The islets are unchanged. The lungs show bronchitis, bronchiectasis and pulmonary abscesses. Atresia of the gastrointestinal, urinary and biliary tracts may be associated with the pancreatic changes. Fatty changes in the liver are found in about ⅓ of cases according to Anderson[7].

4. Dermoid cysts.

5. Teratomatous cysts are rare. They arise higher in the abdomen than omental and mesenteric cysts. X-ray may be very helpful in their diagnosis. Extirpation is the treatment.

Acute Inflammation

Acute inflammation of the pancreas commonly defined as acute pancreatitis represents a varying degree of necrosis of the pancreas, with autodigestion of pancreatic cells, resulting in activation of trypsin and producing a differing clinical manifestation, depending on the degree of insult.

Etiology:—There are generally accepted the following causation factors, but the mechanism of this production of acute pancreatitis is not clearly understood and is still a theorem.

1. *Trauma.* Accidental or postoperative. The increasing incidence of this complication in upper abdominal radical surgery is disturbing and contributes to the mortality, particularly in duodenal gastric and splenic surgery.

2. *Reflux of bile into the pancreatic duct* incident to a common channel, to stone, or edema of the ampulla of Vater or spasm of the Sphincter of Oddi.

3. *Infections* such as lymphogenous or hematogenous spread from distant foci or from the duodenum or biliary system.

4. *Obstruction* in pancreatic ducts as a result of stone, stricture or tumor.

5. *Circulatory* stasis, thrombosis or embolism.

6. A combination of two or more of these factors.

There are certain observations that are pertinent. (a) the trypsin of the pancreas must be activated by some mechanism not clearly understood, (b) pancreatitis is found in middle-aged obese patients, (c) there usually coexists a high incidence of biliary tract disease, and (d) there is frequently an incidence of alcoholism, (e) pancreatitis is not an uncommon cause of acute surgical abdomen, (f) the condition is not commonly diagnosed clinically. Thirty-five per cent are correctly diagnosed, (g) methods of treatment are controversial. If surgery is employed, operative treat-

ment should be properly timed and at best it does not extirpate or eliminate either the disease or its cause.

There are all three types: 1. Acute edematous pancreatitis; 2. acute hemorrhagic necrosis and 3. acute suppurative.

Type 1 may eventuate into Type 2 or 3.

Types 1 and 2 may form a pseudocyst. The common type that may develop into a pseudocyst is Type 2.

Acute Edematous Pancreatitis

This is the commonest of all types. Although recognized by Fitz, Halstead and Archibald, earlier it was established as a distinct clinical entity by Zoepffell[8]. But Elman[9] must be given the credit for its clinical recognition in the United States. The onset is sudden. Pain located in the epigastrium initiates the attack. The pain is in paroxysms radiating to the back, frequently to the shoulders, most commonly, to the left shoulder. The pain is frequently refractory to morphine. There is nausea and vomiting, early abdominal spasm and marked tenderness with distention of the upper abdomen and diminution of, or disappearance of, peristalsis.

The pancreas is enlarged, tense, swollen, soft and boggy. There may be blood tinged or cloudy exudate. Frequently there is fat necrosis localized to the pancreatic region in many cases.

In the majority of cases the urinary and blood amylase values are increased early in the disease. There appears to be a correlation between the concentration of this ferment and the severity of the disease.

Infection of the biliary system, with or without gallstones, is commonly found. This process tends to subside. Repeated attacks produce the condition of chronic intestinal or relapsing pancreatitis.

Needless to say the routine utilization of the serum or urinary diastase test in suspected gallbladder attacks or exploration at operation of the pancreas, will reveal many unsuspected cases.

Conservative management is the proper treatment employing such methods as the control of distention by intubation and Wangensteen suction, relief of pain using demerol, paravertebral injection of procaine for pain and shock, hydration, penicillin, streptomycin, blood, detection of complications such as diabetes, abscess, pseudocysts, etc. and eradication of biliary tract infection after subsidence of an acute attack and adequate preparation of the patient.

If the diagnosis is not clearly established, an operation will be necessary. The operation must consist of the drainage of the lesser peritoneal sac and decompression of the biliary tract by drainage of the gallbladder or drainage of the common duct.

Acute Hemorrhagic Pancreatitis

This condition represents a severe prostrating pancreatitis, characterized by intense, agonizing pain, shock, pallor, subnormal temperature, vomiting and diffuse distention. Classical signs of shock are manifested early. Fluid is demonstrable early in the abdominal cavity. There may be discoloration around the umbilicus;

(Callen's sign[10]). Left flank discoloration, (Grey-Turner's sign[11]) may be evident. Blood examination shows hemoconcentration and diminished blood volume. The serum amylase and serum lipase values are usually increased. There is however no correlation between the severity of the attack and the concentration of these ferments. WBC is elevated showing a preponderance of polymorphonuclears, hyperglycemia and glycosuria may be found. Hypocalcemia has been observed first by Wiper and Miller[12].

Differential Diagnosis

The following conditions must be considered:

1. Acute cholecystitis with perforation.
2. Gastric or duodenal perforations.
3. High intestinal obstruction.
4. Ruptured appendix with spreading peritonitis.
5. Mesenteric thrombosis.
6. Coronary occlusion.
7. Spontaneous rupture of esophagus.

X-rays of the abdomen have a distinct place in the differential diagnosis. They will show the presence or absence of air under the diaphragm in the pleura or mediastinum. They may show dilated loops of the small intestine, isolated dilatation of the transverse colon, (Gabich sign) or fluid in the abdomen.

Among the other aids in diagnosis is serum lipase (Comfort and Ostenberg[13]) which is accurate in 99 per cent of the cases. Splanchnic block procaine (Albanese) renders immediate improvement and relief of pain. According to Elman[14], 60-200 is considered normal for blood amylase, while 200-400 is normal for urinary diastase. There is also the abdominal paracentesis with aspirations of characteristic prune-juice fluid which is almost pathognomonic, and Lowe's Test, the epinephrial eye test, with the positive test suggestive.

Treatment is nonoperative in acute hemorrhagic pancreatitis. General principles of treatment are similar to those in acute edematous pancreatitis, but because of high incidence of shock, blood and blood substitutes must be liberally used. Antibiotics should be given in large doses. Complications of suppuration and pseudocyst formation must be detected and treated appropriately.

Pancreatic Suppuration

Some degree of suppurative inflammation is present in many cases of acute pancreatic necrosis. If the patient survives the acute attack, suppurative inflammation may supervene. In other instances the attack seems to be that of a suppuration process, the acute phase is less dramatic and signs of sepsis are present from the onset. An abscess or multiple abscesses may remain localized; may burrow retroperitoneally to the subphrenic area or down to the left iliac fossa, or rupture into the general peritoneal cavity. Suppurative inflammation should be treated by operative drainage.

TABLE V

ACUTE PANCREATITIS

Nonoperative—11 acute, treated medically, 2 deaths, 18%

Operative—1 traumatic, recovery

9 acute) 2 edematous, no deaths

) 7 hemorrhagic, 1 death, 11%

Total—21 cases

PSEUDOCYSTS OF INFLAMMATORY ORIGIN

Pseudocysts are accumulations of bloody fluid or products of autodigestion and degeneration of the pancreas, occurring in the region of the pancreas as a result of pancreatitis or of trauma. They have no true wall and are limited by adherent adjacent walls. They may occur after a single attack, or occur in patients who have had recurrent attacks. A collection may occur days, weeks or months after an attack. Ferments are found in the cystic fluid in the majority of cases. Symptoms include fullness, distress, epigastric pain, tenderness, anorexia, occasional vomiting, loss of weight and low grade fever. X-ray with barium meal, or X-rays with in-lying tube demonstrated an epigastric mass displacing the stomach, duodenum or large bowel.

TABLE VI

PSEUDOCYSTS

Pseudocysts—7

a. Traumatic—1

b. Acute pancreatitis—3

c. Chronic relapsing—3

8 Operations—Marsupialization

1 Case requiring second drainage of a recurrent cyst

The majority of these cysts are present through the gastrocolic omentum. They are usually present to the left in the midline.

Treatment is operation, marsupialization. A small number may recur. If an accumulation of fluid recurs, a third time, resection of the involved pancreas must be performed. Anastomosis of the cyst to the stomach or duodenum, may be preferable in the recurrent cyst or the cyst with persisting fistula.

CHRONIC RELAPSING PANCREATITIS

This was described as a clinical entity by Comfort et al. in 1946. It is a disease of the pancreas characterized by recurrent attacks of pain in the upper abdomen. During these attacks there is evidence of pancreatic dysfunction. At the onset there is evidence of islet and acinar tissue disturbances, but only during the acute phase. As these attacks recur, evidence of permanent injury becomes apparent. Eventually these changes result from recurring edema, necrosis, hemorrhagia, etc.

This disease is not a new discovery. Neither is it an uncommon one. It may be recognized for the first time presenting one or several signs or symptoms of many possible predominating ones.

It may precede or follow biliary tract diseases. It may follow perforated peptic ulcers of the duodenum or stomach. It may complicate trauma, tumor of the ampulla of Vater, or tumor of pancreas. Alcohol is a contributing factor.

Recurring pancreatitis may produce calcification, fibrosis, cyst formation, steatorrhea, creatorrhea, diabetes, acute or chronic, jaundice, gastrointestinal hemorrhage, calculi, in biliary or pancreatic ducts, cholangitis, hepatitis, splenomegaly, ascites, and drug addiction.

In the acute phase the treatment is medical. It is necessary to search for a possible cause and eradicate it if possible, eliminate biliary tract disease and remove an activating ulcer or any source of focal infection.

In the chronic phase, surgical treatment is indicated. It varies with the individual case and falls under the following headings:

1. Eradicate associated biliary tract disease.
2. Sphincterotomy—Doubilet and Mulholland[16].
3. Pancreatoduodenectomy.
4. Total pancreatectomy.
5. Splanchnecectomy unilateral or bilateral—Reinhoff and Baker[17].

Retention Cysts.
Pancreatic Calcinosis.
Pancreatic Fibrosis.
Pancreatic Duct Calculi.
Pseudocysts (Chronic Inflammatory).

TABLE VII
RELAPSING PANCREATITIS

1 Case had sphincterotomy
3 Cases had sphincterotomy plus cholecystectomy
2 Cases had duodenopancreatectomy
1 Case had common duct drained
1 Case had gallbladder drained
1 Case had exploration, bilateral splanchnicectomy, total pancreatomy
5 Cases explored—no treatment
3 Cases pseudocysts drained
2 Cases untreated—)active duodenal ulcer posterior wall
)chronic gallbladder, probably stones
Total—19 Cases

These conditions can be pathological expressions of relapsing pancreatitis. They are not to be considered as distant entities and are found to coexist in varying degrees. Each case must be individualized and treated according to general principles aimed at correcting, if possible, the presenting disability. Complicating diabetes in these cases tends to be severe, progressive and refractile to treatment.

PANCREATIC TRAUMA

There are two main causes for pancreatic trauma, the operative and the non-operative.

Operative trauma is secondary to excision of a penetrating duodenal ulcer or to procedures on the terminal biliary tract, the stomach or splenectomy. Splenectomy is particularly for trauma. Pancreatitis producing subphrenic irritating accumulations or collections in the vicinity of suture lines may produce necrosis of suture lines with leak and spreading peritonitis. Fortunately, at times, collections may tend to localize and drain through abdominal incisions producing external

fistula. After drainage of intraabdominal collections, external fistula may be troublesome but are usually only temporary, due to communication with a small duct or ducts. Careful drainage of an area of injured pancreas at the time of operation may be indicated. This is a wise procedure in many cases of total gastrectomy when splenectomy is performed.

Nonoperative trauma:—It is good surgery to remember that apart from penetrating wounds, nonpenetrating wounds to the pancreas such as contusions, lacerations and rupture, may produce internal collections, pseudocysts or peritonitis. In the suspected traumatic pseudocyst, operative exploration must not be delayed.

Cyst Adenomas

These are true tumors, proliferative and usually multiocular. They are uncommon and develop insidiously often attaining a large size before they produce signs of deformity, anorexia, nausea, vomiting and weight loss. They usually arise from the body of the pancreas and tend to become adenocarcinomatous. Complete excision, invariably difficult and carrying a considerable mortality is indicated.

Islet Cell Adenoma—Nesidioblastoma

This is the most common of the benign tumors of the pancreas averaging one case in 800 autopsied cases. Only 20 per cent have symptoms. The first case was reported by Wilder and associates[18] in 1927. An adenoma is a small, highly vascularized tumor occurring in the substance of the gland commonly in the tail of the pancreas. They may be single or multiple. The insulin content is 4-40 times the content of normal gland per gram weight.

The symptom complex is that of hypoglycemia, with all of its accompanying features and consequences. The patient is in good health and presents a history of an insidious onset of a chronic progressive episodic course. He has attacks of headache, restlessness, dysarthria, diplopia, tonic or clonic muscle spasm, convulsions, hyperreflexia and occasionally coma and death are expressions of disturbed central nervous system.

The criteria for diagnosis according to Bockus[19] are as follows:

(1) Fasting B. S. below 50 mg. per cent.

(2) Fasting B. S. below 40 mg. per cent following a provocation test.

(3) Relief of attack when glucose is given intravenously without the patient's knowledge.

(4) Classical glucose tolerance curve. Rise not above 120 and fall to subnormal levels persisting for 3-6 hours.

(5) Normal health and stable nervous system between attacks and

(6) Progressive course.

The surgical treatment includes the removal of adenoma or adenomas, resection of pancreas if no adenoma is discovered and resection of the tail of the pancreas.

Carcinoma of Pancreas

1. *Islet cell adenocarcinoma:*—Adenoma that lack encapsulation, microscopic invasion of blood vessels with metastasis. They are uncommon and are found in

the younger age group. The hypoglycemia does not respond to excision of the main tumor because the metastatic tumor nest produces insulin. Surgical treatment is unsatisfactory.

2. *Cyst adenocarcinoma:*—Cyst adenocarcinoma is very rare. It occurs in middle life and is rapidly growing, painful, nontender, upper abdominal mass. There is disability and weight loss. Diagnosis is usually verified at operation by biopsy. They do not lend themselves to radical extirpation.

Sarcomas, Lymphomas

The clinical picture is essentially that of cancer of the pancreas. Their course in time is rapidly progressive. Growth is rapid and the tumor may attain a considerable size and be palpable. *Surgical excision* is invariably impossible due to early extension and rich vascularity and x-ray treatment is unsatisfactory.

Adenocarcinoma of the Pancreas

Cancer of the pancreas occurs usually after age of 50 and occurs in the female in ⅓ of the cases, and ⅔ in males. Two per cent of all cancers occur in the pancreas, ⅔ of all cases occur in the head and the remainder, in the body and tail. The incidence in the ampulla of Vater to pancreas is about 1-12. All ampullary tumors are adenocarcinoma. Three-quarters of the cases of cancer of the pancreas are adenocarcinoma. The remainder are made up largely of duct cell carcinoma.

A painful dull-aching backache, which is more pronounced on lying flat in bed at night is a common symptom of adenocarcinoma of the pancreas. Jaundice is an early prominent feature and it is progressive, present in 80 per cent of cases. There is progressive weight loss and weakness, anorexia, nausea and vomiting coexist. The gallbladder is dilated in 75 per cent of cases. Diarrhea creatorrhea, steatorrhea occur in the majority of cases. X-ray studies may show an enlargement of the head. Hepatomegaly is a common finding and diabetes is present in 13 per cent of the cases. Peripheral thrombosis in 8 per cent. Ulceration of the upper gastrointestinal tract occurs in 20 per cent, while 20 per cent have stones in the gallbladder, 5 per cent have stones in the common duct, pancreatitis is present in 50-plus per cent of the cases, cyst formation is present in 15 per cent, fibrosis is present in 85 per cent and fat necrosis in 23 per cent, confined to the pancreas.

Metastasis occurs in the following order:—(1) Extrahepatic biliary ducts, (2) Liver, (3) Regional lymph, (4) Duodenum.

Accuracy in preoperative diagnosis in the early case is surprisingly low. At autopsy 18 per cent are found to be operable, and 13 per cent curable, with disease limited to the pancreas.

The treatment is surgical. Whipple, Parsons and Mullins[10] demonstrated the feasibility of radical surgery in 1935. Later in 1940, Whipple performed the operation in one stage for the first time. His operation with few modifications has become the standard procedure wherever surgery for malignancy of the pancreas is performed.

Many surgeons have reported *poor* end results. The fault is not in the procedure, but rather in the operator's selection of cases for radical operation. An improved criteria of operability *must be established and taught.*

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GASTROJEJUNOCOLIC FISTULA*

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This late complication of gastric surgery remains one of the most serious in the operative management of gastroduodenal ulceration and fortunately is of infrequent occurrence. Early recognition and proper management are essential if the patient is to be effectively treated. During recent years the incidence appears to have decreased perceptively, but in the light of the present trend in management of peptic ulceration with the performance of more gastroenterostomies with or without vagal resection, an increased incidence may be anticipated. Other causes than gastric surgery for the development of this fistula are to be found in gastric or colonic carcinoma, intestinal tuberculosis, trauma, etc., but these entities are not included in the present consideration.

Pathogenesis and Course

The pathological sequence which leads to a gastrojejunocolic fistula is initiated by stomal or marginal ulceration arising at the site of a gastroenteric anastomosis. The perforation of such an ulcer into the transverse colon to which the ulcer has gained adherence establishes the fistulous communication. While instances occur in which the tract extends only between the jejunum and colon, it is more frequently true that all three viscera are joined. It is extremely unlikely that, once established, any such mucosa lined tract ever undergoes spontaneous closure although it is conceivable that initial perforation with abscess formation and the secondary extension of the abscess into the colon may set the stage for cicatricial contraction and ultimate obliteration of the lumen of the fistula. The usual course is for the fistula to maintain itself, frequently with scarring in the wall of the colon so that significant obstruction of the lumen may occur.

While it is possible for gastric content to pass directly into the colon through the fistula, passage in the reverse direction is evidently the more common course. This is supported by the experience of the roentgenologist whereby the communication is more readily visualized by barium enema than by upper intestinal barium series. As a result of the small bowel contamination, an active jejunitis and ileitis is set up resulting in marked hyperperistalsis and the rapid passage of ingested material through the intestinal tract. Upon this prime effect is based the outstanding clinical feature—the diarrhea and the profound nutritional and biochemical changes which follow.

Incidence

The presence of stomal peptic ulceration serves to indicate that the original ulcer-forming mechanism has not been effectively or permanently altered by whatever surgery was originally carried out. Marginal ulceration of this character

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occurs in 3-4 per cent of cases following gastroenterostomy according to Walters and Clagett[1]. This is a conservative figure as compared with that of 15 per cent advanced by Lahey[2] and still higher figures from other responsible sources[3]. The incidence of marginal ulceration is significantly less in gastrojejunostomy following subtotal gastric resection and this is to be looked upon as a mark of increased efficacy of resection as a method of treatment in duodenal ulceration. It is likewise true that the complication is less frequent in the surgical management of gastric as compared with duodenal ulceration and this is apparently related to the lower degree of hyperacidity in the former lesion. The percentage of marginal ulcers which progress to the formation of gastrojejunocolic fistulae is placed between 11 and 14 per cent[4,5]. Since the total number of gastrojejunocolic fistulae which can be found in the literature is surprisingly small, it must be apparent that this is a figure which is difficult to substantiate, but the various estimates on the part of responsible observers lie essentially within the range indicated. It is significant that this complication rarely occurs after anterior or antecolic gastroenteric anastomosis and this is due to more direct proximity of the anastomosis to the colon in the posterior and not in the anterior type. Marshall[6] calls attention to the fact that when a posterior anastomosis is made the opening in the transverse mesocolon should be close to the base of this structure and therefore contact with the colon is less likely to occur. This of course may set the stage for perforation of a stomal ulcer into the mesocolon or into the pancreas with its own symptom picture, but fistulation into the large bowel is avoided.

As previously indicated the incidence has undergone a considerable drop in recent years and Faxon[12] points out that in the past forty years only 18 cases have occurred at the Massachusetts General Hospital and that none has occurred since 1941. This diminution is undoubtedly due to the general improvement in surgical management which has taken place in all large centers, but particularly to the substitution of adequate gastric resection for posterior gastroenterostomy as a definitive type of surgery. The changing trends of the present period make it probable that gastroenterostomies will be done more frequently in combination with vagal resection and if the latter procedure is incomplete and previous gastric hyperacidity is allowed to recur, the stomal ulceration and its attendant complications are to be anticipated.

Clinical Syndrome

There is a wide variation in the latent interval between primary surgery and the development of the fistula[4,5] and it is important to point out that cases have been reported within the first year and after 40 years. The time factor alone, therefore, does not exclude the diagnostic possibility. Careful questioning will frequently disclose that recurrent gastric symptoms have occupied the period which precedes the actual fistula formation. The ultimate development of the fistula may be quite sudden and without warning or, as is suggested, may have been preceded by the syndrome of marginal ulceration.

Pain:—This is likely to be severe in the period which precedes the development of the fistula and during which the stomal ulcer is active, but once the com-

munication is established spontaneous pain is no longer a prominent or consistent part of the clinical picture. Local tenderness however may be present as a mark of the inflammatory reaction about the fistula.

Diarrhea:—This is the focal point of the symptom picture and is usually severe and debilitating. Watery movements follow the taking of any food or fluid and frequently undigested material may be recognized in the feces.

The diarrhea is less a mark of direct stomach to colon shunt than of the irritative jejunitis and ileitis induced by the regurgitation of colonic content into the stomach and jejunum. This small bowel irritation was strikingly demonstrated in a case under consideration by the marked congestion and edema of the bowel wall; in appearance quite in keeping with that of early regional ileitis.

Stercoraceous Odor to Breath:—The patient gains early awareness of the foul odor which in some instances immediately precedes the onset of the diarrhea. This complaint is partly responsible for the anorexia which contributes to the developing nutritional deficiency.

Weight Loss:—Rapid and continued weight loss is also a constant finding and is a mark both of fluid loss and inanition and suggests the comparable state which develops in acute ulcerative colitis. In the patient reported in this paper the weight loss amounted to 16 pounds within a period of less than five weeks.

Local Findings:—The presence of a tender mass immediately above and to the left of the umbilicus has been reported but is not a constant finding. Tenderness at this site is usually to be defined but does not represent a prominent part of the physical picture.

Nutritional Deficiency:—Atwater[4] and his collaborators call attention to the deficiency states brought about by the rapid passage of solid and liquid food through an altered intestinal tract. Hypoproteinemia results in edema and occasionally in ascites. Iron deficiency is a constant finding and nutritional anemias are frequent. The laboratory evaluation with respect to the total red blood count and hemoglobin may lead to false interpretation due to the hemoconcentration which is present. The specific deficiency states attributable to faulty absorption of the essential vitamins have all been reported.

Roentgen Examination:—X-ray examination by the administration of a barium enema demonstrates the fistula in practically all instances, the opaque medium entering the stomach and small bowel simultaneous with its appearance in the transverse colon. As pointed out by Gray and Sharpe[7] an upper gastrointestinal series is less likely to demonstrate the fistula because of the edematous folds of jejunal mucosa which project into the lumen.

Preoperative Management

While proper surgical management is the only basis on which cure is to be expected, adequate preoperative management is the only basis on which the surgery can succeed. The various nutritional factors mentioned indicate the direction to be taken in the preparation of the patient, but the specific methods used are influenced and greatly modified by our inability to use the alimentary canal to

restore the patient. Jejunostomy for feeding purposes had its proponents in a former era, but it seems unlikely that the jejunum would tolerate material introduced by an adventitious opening any better than that directly introduced by mouth. Moreover the presence of an additional jejunal stoma would unnecessarily complicate the ultimate surgical management. It becomes necessary therefore to utilize the intravenous route for the restoration of body fluid, electrolytes, protein, blood and vitamin stores. The virtual withdrawal of all fluid and food by mouth reduces the stimulus to frequent diarrheal movements and therefore reduces the steady fluid drain. It is necessary to maintain the hygiene of the mouth by the judicious use of oral preparations. Gray and Sharpe[7] report a reduction in operative mortality from 61.5 per cent to 27.7 per cent on the basis of adequate preoperative care alone. Of necessity, from 1-2 weeks will be required to restore nutritional balance and during the latter part of this period the use of streptomycin for intestinal bacteriostasis or the administration of sulfathalidine will reduce the operative hazards.

Surgical Treatment

Surgical treatment must have two objectives—first, the effective interruption of the fistula, and second, the control of the primary ulcer diathesis. Failure to consider the second issue has repeatedly led to the recurrence of the original duodenal ulceration and Faxon[13] cites three instances in which the reformation of the duodenal ulcer occurred within a period of three months to one year. That the objective mentioned is not generally pursued is indicated in presentations of Thomas[9] and Wayland[10]. Thomas reports four cases of gastrojejunocolic fistulae due to ulcer, of which three were treated by taking down the existing gastroenterostomy and reconstituting normal intestinal continuity. In one case of the four a supplementary anterior gastroenterostomy was done. Wayland[10] presents three cases in which intestinal continuity was restored apparently without any effort being made to insure lasting reduction of gastric secretion. Thomas nevertheless quotes from Lahey's experience that—"The unhooking of a gastroenterostomy and restoration of the digestive stream has resulted in a recurrence of the original ulcer in 40 per cent of their cases".

The completion of the full operative program in a single sitting or its division into two or more stages will depend upon many factors, prime among which is the condition of the patient. Various "first stages" have been proposed primarily for the patient so depleted by the intractable diarrhea that full effort at parenteral support and restoration is unavailing. Pfeiffer[11] in 1939 suggested proximal colostomy as a means of preventing small bowel reflux and the adoption of this brought about a striking reduction in over-all mortality.

Garlock[8] and Dreosti[12] have suggested that cecostomy be done instead of right colostomy, primarily to remove the site of the intestinal stoma as far as possible from the field of definitive surgery. The relative effectiveness of the latter procedure is to be questioned but evidently it has served these authors well. Marshall[6] proposed ileodescending colostomy as the first stage and the reported

good results supported his convictions. It must be borne in mind however that the many adjuncts to modern management of major intestinal surgery contribute to the mortality reduction and that the specific procedure itself is not solely responsible.

Various specific programs of surgical management may be outlined.

I. Exposure of the fistulous site with separation of the three adherent viscera and primary closure of the several defects. This constitutes an invitation to reactivation of the original duodenal ulcer unless some attempt is made to combat the ulcer diathesis—the addition of an intraabdominal subdiaphragmatic vagotomy will be a tempting addition to the essentially simple procedure mentioned. The direct invasion of the involved bowel wall carries with it the hazard of peritoneal contamination and questionable security of the multiple suture lines, but the risks will be reduced by the liberal administration of antibiotic preparations.

II. Primary exposure of the fistula with its resection including the adjacent segments of involved bowel,—normal intestinal continuity is then reestablished and supplemented by abdominal vagotomy. This in essence is the procedure advocated by Faxon and Schoch[13]. The program is one for the good risk patient not in need of a preliminary diversion colostomy and calls upon one's conviction that the vagus resection will serve to maintain permanent control over the ulcer tendency.

III. Preliminary diversion of fecal stream by colostomy followed by excision of fistula, closure of jejunum and colon, and gastric resection as recommended by Pfeiffer[11]. This of course requires a third procedure, the closure of the diversion colostomy and in addition requires that the major operative performance be carried out after the abdominal wall has been contaminated by the colostomy. This objection to be sure is readily overcome by careful technical management and the proper use of antibiotics. One technical objection lies in the attachment of the transverse colon to the abdominal wall and the interference which may in a measure complicate the definitive surgery.

IV. The fourth approach is that advocated by Marshall and Lahey[6] and consists of a first stage ileodescending colostomy followed by en bloc resection of right colon, the greater portion of the transverse colon, the involved jejunal segment and the lower three-fourths of the stomach. The preliminary diversion of the fecal stream from the fistula is theoretically the most effective which can be obtained and serves to prepare the site of the fistulous communication for the ultimate surgical resection. The second stage involves the sacrifice of considerably more colon than seems necessary, but technically this is no serious issue and physiologically the organism adjusts very well to the sacrifice. Two instances of spontaneous closure of the fistula after the first stage have been recorded—one by Marshall[6] and the second by Lahey[15].

Case Report

The patient, A.A., is a 60-year old male originally operated 22 years ago for a chronic recurrent duodenal ulcer. At that time a posterior short loop gastroenterostomy was performed with complete relief of the symptom picture. The patient remained well and active until 8 years ago when he was hospitalized and studied for persistent midback pain. While the prime emphasis was placed on orthopedic possibilities and a myelogram was performed to exclude the presence of a protruded intervertebral disc, it seems likely that the pain was a mark of slow penetration of a gastrojejunal ulcer.

In December 1948 the patient became aware of foul eructations and a bad taste in his mouth and shortly thereafter there began a persistent and debilitating diarrhea. The bowel movements were loose and watery and were induced by the taking of any food or fluid. Within 10 days the patient lost approximately 15 pounds and had placed himself on a program of restricted food and fluid intake to reduce his diarrhea. The resultant picture of dehydration was striking.

Upon hospitalization the following laboratory data was obtained: W.B.C. 6,800; R.B.C. 4,650,000; hemoglobin 88 per cent; N.P.N. 20; serum protein 5.5 grams with an A.G. ratio of 1.5. Gastric analysis revealed 30 units of total acid with no free hydrochloric acid. Urine examinations were essentially normal.

X-ray examination by upper gastrointestinal series and by barium enema revealed a gastrojejunocolic fistula.

It was decided because of the extreme changes which had been brought about within a short time to divide the patient's surgery into two stages. Preoperative management continued for 9 days and included the administration of intravenous glucose, saline and amino acid solution, concentrated polyvitamin preparations and transfusions. Streptomycin by mouth was started 3 days before operation and administered in ½ gram doses four times daily.

The first stage operation consisted of ileodescending colostomy without any effort being made to explore the vicinity of the fistula. The distal ileum was turned in and the prime anastomosis established side to side. Of great significance was the startling inflammatory aspect of the small bowel—bearing as it did the suggestion of a diffuse inflammatory process. The small bowel was accorded the respect which is so necessary in ulcerative colitis. The postoperative course was innocent and at the time of discharge on the tenth postoperative day, the patient was having formed stool and the stercoraceous odor to the breath had entirely ceased.

In the interval between operations barium enema examination was twice made in the vain hope that spontaneous closure of fistula might occur. Since it did not, the patient was re-admitted after an interval of five weeks and four days later was reoperated. While the patient had not gained weight, he looked and felt very much better than when originally seen.

The second operation consisted of an en bloc removal of the right and transverse colon approximately to the splenic flexure including the involved jejunum and the distal two-thirds to three-fourths of the stomach. The total inflammatory mass

which surrounded the fistula was of considerable size and would have made any of the direct approach procedures much more difficult than this apparently more radical procedure. The jejunal ulcer and the fistulous communication were readily demonstrable in the surgical specimen. The patient tolerated the surgical effort very well and received supporative transfusions and the intravenous administration of glucose and saline. His convalescence was complicated first, by the development of a severe postoperative parotitis and second, by the development of intense jaundice on his 7th postoperative day. The jaundice was looked upon as an instance of homologous serum jaundice and was related to the previous operation and transfusion carried out at that time. The parotitis responded promptly to radiation therapy.

The patient was discharged much improved on his 16th postoperative day and has since returned to his usual work. His weight has slowly risen approximately to his normal and except for increased frequency of bowel movement there has been no distressing gastrointestinal symptom. There has been no diarrhea.

Summary

I. The pathogenesis, course and incidence of gastrojejunocolic fistula is considered.

II. The clinical syndrome is discussed and the importance of diarrhea as a basis of the profound clinical derangement is stressed.

III. The great importance of preoperative management is emphasized, and various types of operative procedure are described.

IV. A classic case is reported in which a two-stage procedure was followed with complete recovery.

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CORTICODIENCEPHALIC GASTROINTESTINAL SYNDROMES IN EPILEPTICS*

(PART IX)

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HEREDOFAMILIAL HISTORICAL FACTORS

Throughout the presentation of the data on the 300 cases of epilepsy we have repeatedly attempted to impress the reader with the fact that although we were presenting seemingly unrelated material, it was very intimately connected. Thus, we showed that in the extensive presentation of the aurae of the gastrointestinal and nongastrointestinal types, that we were merely giving part of the chief complaint. We did likewise when we gave an elaborate description of the types of seizures and the statistical data connected with them. This we also continued when we gave details on the various types of assigned and other causes as well as the past personal history of the individual. In other words, we were continuously proving that just as in the diagnosis of a case and in the placing of a cerebral lesion in general neurology, we take the chief complaint, the present history, the past personal history and the family history and combine them with the symptomatology and the neurological findings in order to arrive at a conclusion which gives us an anatomical localization of the lesion within the cerebral hemisphere, so in focal and general epilepsy this procedure is indispensable. In other words, speaking in the terms of epilepsy, and particularly in the gastrointestinal type of epilepsy, we place the lesion within the cerebral cortex or below it, that is, within the corticothalamomammillary tract or within various other parts of the cerebral cortex which directly or indirectly connects with the final common pathway, that is, the spinothalamic or the pyramidal tract. The reader can readily see that we hardly digressed from the material which we presented at the beginning of our paper and from that which we repeatedly stressed under the heading of neuroanatomy of the corticodiencephalic system. In the gastrointestinal syndrome groups which we eventually hope to describe, a procedure similar to the above will be followed in each case presentation of the case histories of each group. The aurae dealing with abdominal pain and those with other sensations as well as the motor aurae, will eventually present us with definite anatomical lesions of the corticodiencephalic system that

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will be backed by positive neurological findings pointing to these areas and in some instances by pneumoencephalographic proof. While we presumably work on epilepsy with its anatomic and pathologic features we simultaneously solve the problem of neurogenic control of gastrointestinal function since the gastrointestinal aura is associated with the convulsion.

We will therefore continue with only two more subjects dealing with the 300 cases of epilepsy. These two subjects deal with the family history in great detail so that the problem of congenital and hereditary findings in relation to the causation of epilepsy is cleared up. Subsequent to that we shall dwell only in a limited way upon some of the neurological and other findings as presented upon admission. When the syndrome group cases are presented no elaboration upon hereditary or familial characteristics will be made. Only the term heredity, etc. will be used without further qualification. The autonomic links the heredity of the epilepsy migraine syndrome.

There is an extensive literature on the subject of congenital and particularly on that of hereditary epilepsy. Throughout the past century various investigators and outstanding neurologists have attempted to show that there is a definite hereditary type of epilepsy. In the last two decades, however, investigators have disproved an overwhelming incidence of hereditary diseases causing convulsions by comparing their results in tremendously large numbers of cases with those of such outstanding authorities as Gowers and various others. Lennox and Cobb and particularly the former author have delved into this subject extensively, but at this time we have not the space to offer the reader even a short summary of the data on the subject. We ask the reader, however, to distinguish between the term congenital and hereditary epilepsy as we have previously indicated.

The reader will therefore realize that in the presentation of the family history both of the maternal and paternal branches, we will find a great deal of substance which well covers pathological material that might fall under the heading of congenital and hereditary character and thus be related to the lesion within the brain of the epileptic patient in an etiological way, and be secondarily responsible for his epileptic seizures.

The incidence of these familial and hereditary diseases or conditions in 300 cases of epilepsy under consideration as they occurred in the mother, we will present whenever possible according to system. In considering nervous and mental ailments which occurred on the maternal side we find that nervousness, death due to a stroke below 40 years of age, hysterical spasms, fainting spells as a baby, death due to apoplexy, epilepsy, insomnia and depression, death due to paralysis, chorea at 30 years of age, immoral epileptics, fainting spells during pregnancy, nervous temperament, spinal injury causing death, neurotic, over-sexedness and irresponsibility, insanity, death due to fracture of the skull at the age of 48, hemiplegia at the age of 46 years, feeblemindedness, postpartum fainting spells, and finally fainting spells during pregnancy were the diseases and conditions found that seemed to have a possible relationship to the epilepsy of the children when such conditions as enu-

merated above occurred in some of the mothers of the 300 epileptic institutional patients. It is again interesting to recall the relationship between pyramidal and extrapyramidal hemiplegia even in heredofamilial diseases. In degenerative hereditary diseases such as Friedrichs' ataxia, etc. one can readily point to cerebellar factors because it is a spinal form of cerebellar disease and the ataxia is therefore a prominent feature. In corticocerebellar disease however, balance is not so much interfered with and if a hemiplegia is present, although it is associated with corticocerebellar fibre involvement, the paralysis overshadows coordinative disturbances. We are certain that Muskens who did much more than Davison and Goodhart to interrelate the cranial nerve nuclei with Deiters' and vestibular nuclei would appreciate our point of view of relations between pyramidal and hereditary extrapyramidal diseases (Patterson and Weingrow 1937). Other diseases found on the maternal side of the family of the 300 cases that seemed to suggest involvement of the cardiovascular and respiratory systems were the following: death due to pneumonia, to bronchial asphyxiation, to hypertension at 53 years of age, and finally death following unspecified heart trouble. The renal diseases which occurred in mothers of the 300 epileptic patients were few in number. It should be remembered that such conditions, when present during pregnancy in the mother will definitely influence the brain of the offspring. In one case we find that a kidney condition which made the mother bedridden for three months prior to the birth of the patient was present. In another instance death due to Bright's disease was mentioned. Systematically unassigned items such as "parents were second cousins", "died of cancer", "syphilitic", "died of high blood pressure at 53 years of age", "mother died of rupture", "mother was of dwarf type", and a "death due to anemia and blood poisoning" were the other items mentioned in the maternal history of 300 cases of epilepsy. A maternal death due to diabetes, and another one due to gout and diabetes, and a third in which death was due to gallstones were also mentioned. Data such as the above will be meant whenever no specific statement is given and only the words "positive maternal hereditary history" are used. Brief terms such as the above will be employed in the case presentation of the gastrointestinal syndrome groups when other features will be emphasized.

Next in incidence to the maternal conditions involving the nervous system came the genital tract. In this respect the reader will realize that the maternal history is by far more important than the paternal. It is in diseases incidental to pregnancy, those of the puerpurium, and otherwise that one finds a common cause for the congenital encephalopathies and congenital encephalidities and, possibly, even for the congenital malformations of the meninges and brain. In most of our publications we have emphasized that congenital defect was almost as frequent as birth injury and sometimes more so, as a causation of epilepsy. Through the hemic route as well as by direct infection from the womb and adnexae, the fetus can easily contract not only diseases such as syphilis, but almost every infection in the medical calendar can involve the newborn and thus be a responsible factor in the production of pathological conditions which might affect the cerebral cortex and

deeper structures. Not only infectious, but also traumatic, toxic, metabolic, endocrine and heredofamilial as well as mechanical conditions might be responsible for various deformities, and injuries to the brain. The reader should therefore remember that not only the corticothalamomammillary system but particularly the extrapyramidal, (as may be seen in hereditary chorea), pyramidal, and spinothalamic fibres of the final common pathway may be involved, causing not only congenital athetosis, encephalitis and all varieties of hemiplegia but also convulsions with aurae of a specific type referring to the gastrointestinal tract and to the autonomic, among others. Not only are the convulsive manifestations of the brain involvement present, but as we have seen from the work of Davison and Goodhart as well as from that of Archambault and Fromm, that various paralyses and motor disturbances of the eye, head, neck and body scars may be seen in addition to total or partial trophic changes in the body musculature. It is also likely that these are responsible for abnormal head movements in convulsions. From the above we can see how important all possible available data in the taking of a history really are. Items which might seem irrelevant, will crop up now and then and assert themselves to be the causative factors in the production of pathological conditions which are situated in the brain particularly in the cerebral portion and which will produce most of the symptomatology in the mammillothalamic and spinothalamic areas which we have continuously been guilty of repeating and reiterating. With our case presentations the reader will have to keep referring to our neuroanatomical data.

Death in childbirth, a kidney condition which confined the mother to bed three months prior to the birth of the patient, fainting spells during pregnancy, puerperal fever at 36 years of age, postpartum fainting spells, fainting spells when pregnant, and finally cancer of the uterus and breast, were given as the maternal conditions present within the genital tract of some of the mothers of the 300 cases of epilepsy. We must again offer reasons for concentrating upon the past histories of these three hundred nongastrointestinal epileptic cases. There will be certain features which will be neglected when we start dealing with our gastrointestinal syndrome cases. We intend to omit as much as possible group history records because we must concentrate upon neurological, encephalographic and liver percussion in the gastrointestinal cases. The concentration upon neurological findings will have a double purpose. First, it will establish the localization of the neurogenic gastrointestinal corticodiencephalic tracts, a feat, which is the main purpose of this rather elaborate clinical investigation. Secondly, we wish to stress certain parts of the brain such as the cerebrocerebellar pathways which have been neglected not only in epilepsy but in neurology as well. Such pathways are important in conditions such as Menière's vomiting vertigo syndrome.

Although less important than the history of the mother of the epileptic, that of the father should never be omitted in the analysis of congenital and hereditary conditions. Alcoholism and other conditions besides syphilis are factors in the production of epilepsy in the offspring. Data dealing with diseased conditions in the fathers of some of the 300 cases of epilepsy analyzed as far as the nervous system

was concerned are: nervousness, alcoholism, shellshock, epileptic admitted to Skillman in 1930, inflammation of the lining of the brain, delirium tremens, suicide, St. Vitus dance, arrest for indecent exposure, spells, death due to apoplexy, insane, death due to meningococcus meningitis, death due to paralysis, neurotic, immorality, confinement to prison because of assault, hysteria, queer makeup, convulsions all his life, suffered from severe headaches, died at the age of 46 due to sleeping sickness, died at 44 years of age because of "water in his head", paranoid in a veterans hospital, and finally a death due to brain tumor. Other conditions mentioned were: pneumonia at 53 years of age, death due to tuberculosis, death due to laryngeal tuberculosis, death due to influenza, and another death from the same cause, and finally a death due to pulmonary edema, all of which were found in the history of the father suggesting involvement of the lungs. There seems to be plenty of material in the above for those of the modern school of epileptologists who would interpret the intercerebral synchronous diencephalic like dysrhythmias commonly known as the essential epilepsies on a congenital, familial or even hereditary basis. For that matter there is plenty of material in epilepsy for everybody, even for the neurologist as has been shown by no less a great neurologist than Gowers and the greatest neurophysiologist of epilepsy, i.e., Muskens. We will show that Lennox was right, when he attempted to stress the hereditary features of epilepsy on a statistical basis. We intend to do so without overburdening our crowded bibliography. While doing that, we will add authoritative data on the corticopontine and frontobulbar and cerebellar tracts that mediate head movements in the Penfield convulsive patterns. Diseases affecting the cardiovascular system of the father of these epileptics were: diabetic gangrene, cardiac, internal hemorrhage, heart attack, coronary occlusion, gangrene, pernicious anemia and finally Raynaud's disease. Purely heredofamilial direct connections will be dealt with exclusively in the eventual case presentations.

The paternal, metabolic and other gastrointestinal conditions were given as diabetes, death due to rupture of appendix, death following intestinal obstruction, death due to carcinoma of the liver, death due to painter's colic, death due to cancer of the stomach, death following peritonitis, death due to gastroenteritis, death due to duodenal ulcer, another death due to acute appendix, and finally a death caused by liver operations. The renal diseases which occurred in some of the paternal parents of the 300 epileptic patients were: death following dropsy at 70 years of age, death due to arteriosclerosis and kidney disease, death due to tumor of the kidney, death resulting from interstitial nephritis, another death terminating in dropsy at 46 years of age, another death due to nephritis. Were we to fully analyze the incidence of ulcer and gastroenteritis, etc. in the family and past personal histories of the 5,000 cases or even of the 300 cases, as Lennox and Cobb did in their thorough analysis of the literature this report would entail a number of volumes at least. We are merely interested in the neurogenic angle. We wish to establish the fact that if an epileptic patient suffers from abdominal complaints referring to the gastrointestinal tract, it does not mean that the seat of causative pathology is in the abdomen at all times. We feel, that in a certain percentage of such cases, the origin of such

trouble is neurogenic and that if the abdominal spasms are to be stopped, the neural structures causing them should be reckoned with. General conditions which involve some of the fathers of the 300 cases analyzed were given as death due to gunshot wound at 44 years of age, also death due to malaria, and cancer unspecified, death due to syphilis, death due to internal hemorrhage, death following a fall from a building, death resulting from invalid at 73 years of age, and finally a death due to gangrene unspecified.

In the maternal grandmothers' history only one individual with fainting spells was presented. In the paternal grandmothers of the 300 epileptics one had Bright's disease, and another suffered from difficulty in concentration of attention. Five of the maternal aunts showed some conditions, while on the paternal side only two presented findings. In the former there was a history of epilepsy, suicide at the age of 18 years, hysteria, insanity due to alcoholism, mental disorder, and incorrigibility. Of the two paternal aunts who presented some findings, one died of Bright's disease while another suffered from epilepsy. The paternal uncles of some of these patients showed epilepsy and imbecility in one, suicide in another, confinement to the Trenton State Hospital in a third, tuberculosis in a fourth, and finally, alcoholism in a fifth. The sister's child of one epileptic of the 300 cases also suffered from epilepsy while the son of one paternal uncle suffered from a spinal ailment and the child of a paternal aunt also had epileptic attacks. In our gastrointestinal syndrome cases no tracing through generations will be indulged in. If familial disease will be mentioned as a probable cause it will be taken for granted that the direct or indirect effects of syphilis, tuberculosis, genital disease of the mother and alcoholism as well as insanity in the father, etc., etc., are referred to. If symptoms in addition to diseases had been recorded, migraine, and other types of headache, etc., would be added to the foregoing lists.

One paternal cousin suffered from epilepsy, while of a number of maternal cousins, one died of epilepsy at the Skillman Institution, another first cousin had spells, and two other cousins of different epileptics taken from the 300 patients, also suffered from epileptic attacks. The following conditions were found in some of the brothers of the 300 epileptic patients: tuberculosis, death due to convulsions, death following infantile paralysis, death from marasmus at 6 months of age, suicide, death due to being crippled in the legs, incorrigibility and worthlessness, feeblemindedness, epilepsy, death at one day of age caused by enlargement of the thymus, death at 52 years of age due to nervous prostration, tubercular(?), deaf mutism, and finally alcoholism. The paternal brother of one of the 300 patients suffered from fits while the records of the maternal sisters of three patients showed chorea in one, epilepsy in a second, and insanity in a third.

In the sisters of 19 of the 300 epileptic patients the following conditions were recorded: epilepsy, feeblemindedness, mental defect with lisping and stuttering in all sisters, death due to a convulsion, suicide, illegitimate and tubercular, community problem, death due to galloping consumption, death due to cancer of the breast, nervousness associated with crying spells, death preceded by insanity,

syphilis, death from spinal meningitis, death due to sleeping sickness, death caused by marasmus, death due to goiter, deaf mutism, invalidity, and finally fainting spells. Although sleeping sickness is mentioned, extrapyramidal hereditary diseases, such as Huntington's Chorea, were noted to occur in almost no epileptic families, although Tay-Sachs and other forms of amaurotic family idiocies including Schilder's disease were seen. For this reason, even in our gastrointestinal aura cases we were so outspoken in our position against Watts and Fulton's stressing of the extrapyramidal pathways. There seems to be only one avenue by which these experimentalists can extricate themselves from the extrapyramidal snare when so much clinical data is against them. They can claim, and rightly so, that the areas which they stimulated did not produce extrapyramidal movements nor convulsions but gastrointestinal reactions alone, and that they were only interested in these. Only the extrapyramidal syndrome of athetosis is found with both gastrointestinal phenomena and convulsions in the same individuals.

In the paternal grandfathers of five of the 300 epileptic patients, epilepsy was found in one, a second died of tuberculosis, a third was an alcoholic, while a fourth committed suicide, and a fifth was insane. Four of the maternal grandfathers of the 300 epileptics showed the findings of neurosis, alcoholism, tuberculosis, and finally death due to a stroke. It should be pointed out that in the above rather distant relatives, although a strenuous effort was made to contact all the possible members of the family tree, it is conceivable that in a number of cases conditions interfered with accomplishing this. The above material illustrates the differences between contagious as well as infectious familial diseases as seen in the brothers and sisters of epileptics and the purely heredofamilial conditions such as Huntington's chorea, Thomsen's disease, Tay-Sachs disease, Schilder's and similar amaurotic diseases, hepatolenticular conditions, etc., etc., etc., which can be observed in even distant relatives and in which no direct contact such as living in the same house is necessary for transmission of the disease.

Finally, in the family histories of the 300 epileptics we note that in seven individuals out of so many cases, findings pointing to involvement of the nervous system in the patient's maternal grandmother were recorded. A similar number of items were also observed in the patient's paternal grandmother. In the former instance one great grandmother died at the age of 73 from cancer, another had fainting spells, a third suffered from tuberculosis, a fourth was an epileptic, a fifth suffered from an enlarged thyroid gland, a sixth died of a stroke, and finally a seventh suffered from very severe headaches. In the instances of the fathers' grandmother the following were the findings in seven of the 300 epileptic patients: one died of tuberculosis, a second suffered from insanity, a third was a suicide at the age of 30 years, a fourth suffered from syphilis, a fifth also had the above ailment, a sixth was a diabetic at 65 years of age, and a seventh suffered from very severe headaches throughout her life. On a previous occasion we remarked about the various factors which influenced the progress in epilepsy during the last fifty years. The discovery of the x-ray, the isolation of the tubercle bacillus and of the treponema pallida as

well as the clinical descriptions of the various forms of hereditary nervous and mental diseases, all played a part in the evolution of the actual causes of symptomatic forms of epilepsy. In the above are included alcohol and brain trauma. We know that the reader is familiar with such influences and he can gather from the admission case history records, which were collected in the past fifty years, that the march of progress was mirrored even in the wording of the assigned causes. For this reason we made no changes in the data.

Before embarking upon the neurological findings of some of the 300 patients we wish to emphasize a number of points. In our efforts on behalf of the neurology of epilepsy in over a decade of joint activity and publication we feel that we have invariably overstressed the findings in the pyramidal and spinothalamic tracts as compared with those in the corticothalamomammillary and related systems. In all of our publications to which the reader has been referred we have repeatedly presented numerous findings in the former system and in doing so have stressed the excellent work of Henry Head upon cortical neurology. In doing the above we feel that although we have constantly referred to the emphasis by Muskens, Gowers and Brown-Sequard who stressed segmental sensory changes, and various other features found in the routine neurological examinations of patients, we might have been neglecting the diencephalic regions. We have also referred repeatedly to the excellent work of Hodskins and Yakovlev upon neurosomatic deterioration in epilepsy. At this point we haven't the space to refer to the details of extensive elaborations of the above fields as we did particularly in our special paper (Pigott, Weingrow and Fitch 1939 to 1940). In spite of the fact we were disappointed with the setback that epilepsy suffered because of the failure of some epileptologists to pay attention to certain neurological features in epilepsy, we nevertheless feel guilty ourselves because of our own neglect along certain of the above lines. We have done a gross injustice to the frontoparietotemporooccipitopontine pathways. The connection between these and the red nucleus and cerebellum presents an anatomical problem in which tremendous progress has been made in the last two decades particularly through the efforts of Mussen, Larsell, Reilly and others who stressed the ideas of Meynert, Munk and Ziehen. We realize that we might have connected our clinical data of the past ten years with those of the above authors. Of particular relevance is the relation of the subthalamus and other parts of the diencephalon to the tracts involved in the crossed variety of corticocerebellar hemiatrophy. In presenting our cases we will therefore stress this without burdening our bibliography and without straying from the scheme of backing the physiologic basis of corticodiencephalic function with clinical data.

We must confess that throughout this ten year period we have taken our material from the literature and repeatedly referred to the lack of neurological findings, after obtaining our data from the summaries of Lennox and Cobb (1929). Throughout that time, however, we knew, that besides the work of Gowers and others to whom we referred, there was a wealth of clinical neurologic material in epilepsy. True enough, this material cannot always be classed under the heading

of idiopathic epilepsy, and, for this reason Lennox and Cobb desisted from referring to this literature. In reference to the above we have particularly in mind the work of L. Pierce Clark (1900). In his monograph on the subject of clinical studies in epilepsy, Clark discusses exhaustion paralysis in epilepsy, paramyoclonus, multiplex associated with epilepsy, and hypertrophic infantile cerebral palsy and phocomelus associated with chronic convulsions. The last mentioned subject we will present indirectly when we offer the neurological findings of the 300 cases of epilepsy. As far as this subject is concerned we can state that we have elaborately referred to it in past publications. It is the first subject of the above list that is, exhaustion paralysis in epilepsy, that we feel rather especially guilty about. It is a subject concerned with the problem of tonus, neurophysiology, electrophysiology and their relations to the phenomena of motions and problems dealing with the tonic and clonic features of the epileptic attack. In our experience with, and particularly in going over the record of 5,000 epileptics, we have noticed this anatomically and physiologically puzzling phenomenon of exhaustion paralysis rather often. There are many similar phenomena in epilepsy which need interpretation. Proper explanation of most of these depends upon a practical knowledge of neuroanatomy and neuroembryology. Although we have in this paper offered an extensive neuroanatomical literature particularly taken from Kappers we did not delve into the theories of the outgrowth of the neurone. Every neuroanatomist is familiar with the chemical, electrical and mechanical theories of the modern neuroembryologists and particularly with the work of Cajal, Kappers, His, Harrison, Weiss, Detweiler and Coghill. By their experiments with explants they have established the neurone as the unit. Their ideas apply to the anatomy which we presented since our constructions involve nerve tracts. We credited Rosett with the neurotopographic bicellular concept. It may be that such a concept, simple though it be, might explain why transplanted extremities or parts of the same continue growing because of a concentrated central homogeneous topographically bicellular series of tracts subserving all functions. It should, because of the presentation of the nervous system as a progressively organized topographically paucocellular structure, simplify neurophysiological concepts. Fatigue as connected with acetylcholine and other chemical concentrations can therefore be looked upon through the eyes of the cytologist and experimental bioelectric neurophysiologist.

We feel that this exhaustion paralysis and similar phenomena might be classed as belonging to the corticothalamomammillomesencephalic system just as much as to the final common pathway and thus be subject to chemical and electrical influences. That symptoms similar to this condition occur in the syndromes which we intend to present with our gastrointestinal cases may be seen in the following. Manifestation of paralysis in both extremities or contractions in the musculature of the same before and after attacks, whether the same are accompanied by autonomic manifestations or not, whether the same have generalized or Jacksonian seizures or not, has been noted. Not only paralysis of one extremity but of two or three, or

tightening of facial or abdominal muscles has been noted to occur in relation to the epileptic attack rather frequently in gastrointestinal syndrome cases.

If the reader refers to the list of consecutive aurae which we presented previously, he will find the following isolated but definitely focal symptoms preceding the seizure, which, though not exactly motor or paralytic, are nevertheless important in the aforementioned connection. Thus, (35)-gastric and cry, (77)-choking and feeling of pin and needle sensation in the right hand, (98)-choking sensation, (110) (112)-choking sensation, (124)-grinding of the teeth, (132)-nervous sensation about the lips, (177)-chewing of the mouth, (196)-choking in the throat, all refer to unilateral or bilateral focal reactions. The above data, although indicating that there is definite evidence of bilateral activity of certain musculatures which cannot be localized in one side, must nevertheless have a definite representation particularly since they are not simultaneous with the convulsion. There are, however, other evidences of not only bilateral but particularly unilateral phenomena such as paralysis after a fit. These, when associated with a convulsion and particularly when the same is preceded by an aura pointing to the gastrointestinal tract, are significant since they involve the problem of tonus and certain physiological principles which have as yet not been cleared up, although epileptologists have had their interests drawn in that direction. The following gastrointestinal aurae contain such phenomena, and we refer the reader to a continuation of our list in the following: (202)-asks for water and paralysis partial of the left arm is associated with the convulsion, (254)-drawing of the mouth and numbness, (270)-pains in the stomach and the muscles of the stomach contract, also pain in the chest, (295)-tightening and jumping of nerves throughout the stomach and chest. We could have selected many other cases dealing with the subject of exhaustion paralysis as taken from our collection of 5,000 cases but we wanted to confine ourselves to gastrointestinal material exclusively since the case descriptions of the syndrome groups will shed some light on the subject.

In our anatomical descriptions we dwelt at length upon the mammillothalamic system. We took the pains of giving a bird's-eye view of this system to show that its floor in particular contains a great many structures in which there are fibres crossing from one cerebral hemisphere to another. Although we repeatedly hovered between polygon, polyhedron, body of the basket, its handle and tripod, we were essentially anxious to emphasize the importance of the diencephalon as a continuation of the brain stem, as an interhemispheric structure and particularly as a center for the modification of autonomic afferent and efferent impulses. Whether it has a great deal to do with the tonic phase of the convulsion or not we are reticent in committing ourselves. The diencephalon is however the seat of the largest autonomic ganglion, i.e., the thalamus and as such it may be responsible for fatigue phenomena, somnolence, body temperature and (as Stavraky so ably championed) the regulation of the entire cardiovascular circulatory mechanism. We emphasized the anatomy of the base of the brain because for many years we have been observing cases of paralyses in the lower extremities in the form of paraplegia associated

with convulsions. We felt that there must be some other structure in the brain than the corpus callosum which would produce such a condition. Anyone with even a slight neurological training will see that a region confined to one cerebral hemisphere will not produce a paraplegia in the present scheme of neuroanatomic basis of clinical examination. The diencephalic and telencephalic regions at the base of the cerebrum have suffered a peculiar injustice particularly at the hands of some textbook writers. The red nucleus of the genus homo has invariably been placed in the mesencephalic region although it is well known that part of it belongs in the basis cerebri. Decussational and commissural fibres in that vicinity could easily give crossed paralysis and even a paraplegic syndrome. Another structure which might give bilateral motor disturbances is the anterior commissure. It is situated in front of the third ventricle and comes in close contact with the preopticomammillary pathways. Over fifty per cent of our pneumoencephalographed patients (300) show a dilated third ventricle. This could produce many syndrome combinations in a region so somatotopic as the diencephalon. Aside from the paraplegias which we saw we also invariably noticed cases of crossed paralyses which were not of the mesencephalic type. For this reason we felt that the diencephalon, because of its commissural and decussational contents, could produce such combinations of lesions and the patient would at the same time suffer from grand mal epileptic attacks in addition to gastrointestinal symptoms. We therefore presented a detailed quotation from Ingram dealing with the mammillary peduncle. Although for many years we have had inclinations to pick some of these syndromes and describe them in the literature, we have been too busy with other problems in epilepsy. If such lesions, or sets of lesions occur, it stands to reason that a paraplegia characterized as an exhaustion paralysis could be due to some physiological change in the diencephalon or in a portion of the corticothalamomammillomesencephalic system. What has been said above in regard to paralysis can also be said in regard to the segmental sensory changes which have been repeatedly reported in the literature and emphasized by such outstanding authorities as Gowers, Brown-Sequard, Oppenheim, Muskens and many others. For those who abhor to hear that anything in the way of positive neurological findings have been observed in idiopathic cases it might be well to add that the above authorities did find such positive clinical material in the so-called essential epileptic. A lot more could be considered under that heading if postparoxysmal positive Babinski and other reflexes are added.

Since this problem of exhaustion paralysis is so common in epilepsy, and particularly since, so many functions associated with the gastrointestinal tract such as chewing, swallowing, urination, defecation, somnolence, salivation point towards an involvement of the corticothalamomammillary system instead of the pyramidal system, we feel that the diencephalon, particularly its floor, has a definite somatotopic localization. This has been shown to be true by Sherrington and other workers who feel that tonus is located in this region and that in respect to this function of tonus the entire body is represented. We can therefore see that the bioelectric as well as biochemical phenomena associated with fatigue incidental to exhaustion states

could be the agents responsible for a paralysis of the hypertonic and hypotonic types. Action potentials, nerve conduction phenomena, etc., are dependent upon blood concentrations of acetylcholine. Motion and muscle strength are dependent upon electrical activity particularly in cellular ganglion elements which are abundant in the diencephalon. This would mean that chemical and electrical bioneural changes could produce paralysis on the basis of fatigue only if this phenomenon is due to it. It is however unreasonable to assume that only a few jerking activities would induce a focal fatigue lasting days. Only in familial periodic paralysis, and in multiple sclerosis, as well as in myasthenia gravis, is this phenomenon encountered, but only in the first condition does it seem to be clear cut. In none of the above diseases does it follow a convulsive episode. That hypertonicity or hypotonicity, manifesting themselves in the form of paralyses of various nerves, would follow an epileptic attack, because even of a small lesion within the corticothalamomammillary system particularly in mammillary portions and associated localities, is evident. We purposely steer away from the hybrid term palsy which means nothing but which is still used in some circles to mean anything and everything. That such paralytic phenomena could also be accompanied by isolated cranial nerve involvement is logical in view of the fact that fibres extend from the nuclei of the cranial nerve within the brain stem to the floor of the diencephalon. One of us (Weingrow), who has favored the extension of cranial nerve representation into the diencephalon and has done some work on head movements in general, feels that the diencephalon is just as important as the brain stem in the above respects. In all our previous publications we concentrated upon the above ideas in the neurological examinations of epileptic patients suffering from corticodiencephalic and diencephalocortical lesions. We invariably emphasized the teachings of Henry Head on sensory fatigue and the influence of the thalamus in that respect. One of us (Weingrow) claims that, in the few cases of myasthenia gravis that he has seen, he noticed sensory fatigue in addition to motor fatigue. Such ideas, revolutionary though they be, seem to point in the direction of Rosett, i.e., that fundamentally there is little difference between motion and sensation. Maybe the central excitatory state consists of a bioelectric fluid medium that is common to sensation and motion and the thalamus is one of its sources, particularly since it seems to be connected with almost everything that goes on in the nervous system.

To save further elaboration upon this subject of exhaustion paralysis and paraplegia of diencephalic origin we will quote directly from the work of L. P. Clark (1900) and we leave it to the reader who is interested in following this problem to use the references that this authority presents on this most interesting subject. The reader will see that this subject is intimately connected with the paralysis which we will subsequently enumerate in association with the 300 cases of epilepsy: the first quotation from Clark follows: "It would appear that Audry, Thouret and Saillaut, in the 18th century, had noticed the phenomenon of transitory paralysis following epilepsy. The data are not acceptable either in the original or at second hand. A third observer, Perebroom, records the same fact in connection with puerperal

eclampsia in his own wife. These facts were not known by Bravais. Coming to the present century we find a case recorded as early as 1818 by Blaud, several years before Bravais' work was published. No neurologist appears to have noticed that that term *epilepsie hemiplegique* used by Bravais, has no reference to paralysis but simply denotes the lateral character of the convulsion. Todd, on the other hand, in his term *hemiplegic epilepsy*, refers to that form of convulsion followed by paralysis. This discrepancy has produced no little confusion in the mind of subsequent writers." It is unfortunate that L. P. Clark who had written so ably and extensively on the psychic side of epilepsy did not do likewise on the neurological findings in this field. He might have expended the ideas on neurosomatic deterioration in epilepsy. There is just as much clinical data in the last mentioned as in exhaustion paralysis phenomena. Neurosomatic deterioration presents problems that are combinations of Parkinson rigidity without tremor, pseudobulbar symptoms as well as those which one encounters in aphasia. The psychologist and some psychiatrists observing the neurological picture of neurosomatic deterioration are often so perplexed by the above combinations that they not infrequently label as mentally deficient a good percentage of the patients who don't respond because of aphasic and fatigue states.

The following quotation from Clark continues with the historical data on the subject " . . . but Bravais was well aware of the paralysis as an accident in unilateral epilepsy, and records at least one case (cited by Dutil)." After showing that Dutil was the second observer to record this condition, Clark continues as follows: " . . . although Todd taught his views much earlier, they were not published until later. During this 6th decade Russell in England and Robertson in Scotland each reported several cases, and the latter professed allegiance to the ideas of Todd. In France about this time Fere was in charge of the Salpetrière and accordingly Fere recorded temporary paraplegia after epilepsy, but the date and reference are not given. In America during the sixties Echeverria was aware of postepileptic temporary paralysis and there is every reason to believe that he was an independent authority because he had before 1870 (date of his work) kept records of 306 cases of paralysis in epilepsy, six of which were transitory."

Clark continues with his historical data on the subject of transitory or exhaustion paralysis " . . . in France, Charcot or rather his junior, Pitres, was equally industrious in observing and collecting cases, and in making experiments which appear to prove the exhaustion theory. In Germany, Hitzig and Griesinger reported isolated cases. (An outstanding feature about exhaustion paralysis is that in most instances there is no exhaustion connected with it nor is there enough exertion to produce fatigue. We are therefore dealing with periodic, temporary, postparoxysmal paralysis of a focal type. It might even be called a Bravais or Jacksonian paralysis. The periodicity features gives it, if it is not a fatigue phenomenon, a diencephalic characteristic. Periodic phenomena be they migraine, menstruation, coma due to meningeal bleeding or the periodic clinical signs of multiple sclerosis seem to have an autonomic neurogenic factor. It seems therefore not unlikely that the autonomic

operates in this exhaustion focal paralysis either by a local hyperemia ischemia or by a cerebrospinal fluid collection.)

"At the beginning of the eighties Jackson wrote his elaborate theoretical paper which probably gave considerable impetus to the study of the subject. Gowers gave much attention to the postparoxysmal form in epilepsy and took exception to Jackson's (relative diffusion) theory; he also introduced the idea of inhibitory paralysis to explain attacks where paralysis appears to replace convulsions."

With regard to the possible causation of the paralysis that follows a convulsion, Clark presents the following: "Jaccoud as far back as 1864 decided that all paralysis is essentially exhaustive, dependent upon overexcitation. He cites experiments which cover all the ground excepting that afterwards determined by Franck (superexcitation of the cortex), that is, he showed that electric excitation of all nervous tissue, aside from the cerebrum, will produce exhaustion, respectively of vasomotor nerves, peripheral nerves, spinal centers. Hence it would seem that experimentally there is nothing more to be done. However, Jaccoud had especially in mind reflex paraplegia, from the clinical standpoint, and does not apply this theory to subjects like epileptic temporary paralysis."

Clark elaborated upon the subject of hysteria and gave extensive references to the literature dealing with it as a factor in the evolution of this paralysis. In another place in his monograph he dwells at length on the subject of periodic paralysis in relation to the phenomenon of exhaustion.

Whether Clark knew that in mentioning the term *periodic paralysis* he was touching on a neurologic hornet's nest and brought upon himself the invitation to explain a number of other neurologic syndromes, is not evident. It may be that Clark was rather wary about the differential diagnosis of neurologic data such as paralysis in epileptics. Every epileptologist has a tendency to be cautious when dealing with clinical neurologic material in epileptics, fearing, that this might cause a furore among workers who are fond of terms such as idiopathic, symptomatic, essential, etc., etc., and that he might be accused of harboring a notion of solving all etiologies of epilepsy by introducing heretofore unaccepted clinical neurologic findings. We have shown this to be the case with positive findings in all realms of neurologic routine tests in our cases but we invariably fortified ourselves with references to the literature. It is not clear whether Clark meant to connect exhaustion paralysis with the familiar condition of familial periodic paralysis. At any rate, he was not clear on this subject. If, however, both of these conditions are interrelated they could be construed as being produced by some physiological or pathological change in the corticomammillary and periventricular systems. Many other similar conditions and diseases that have gone for centuries without a proper localization within the cerebrum could be explained by anatomical and physiological data such as we have been laboring with in our detailed descriptions of the anatomical dysmorphous polyhedron when we offered a bird's-eye view description of the diencephalic and telencephalic portions of the cerebrum which is intimately connected with the corticothalamomammillomesencephalic pathway. Not only such conditions

but diseases like myasthenia gravis and similar syndromes associated with encephalitis could be connected with such a combination of theories that Clark merely tripped upon, not realizing that he was touching off a spark that might lead to a great neurological conflagration. In Figure 6 (Fitch, Pigott and Weingrow 1939) we see a myasthenic-like facies in an epileptic. The last mentioned author recently performed a pneumoencephalogram upon one of his private cases which was questionably diagnosed myasthenia gravis in which there was evidence of possible involvement of the diencephalon in addition to possible changes in the cingulate and other portions of the cerebral cortical autonomic. He does not advise that this test be repeated too often in cases of myasthenia and in individuals with bulbar paralysis because the cardiac musculature is hardly amenable to strains upon it in the course of the procedure. The above digression teaches us however that the diencephalon of epileptics as seen in the above illustration and in others holds some hope of lesion localization. In Figure 8 of the paper referred to above we see a patient with paralysis of all extraocular muscles and epilepsy and in spite of the above presenting a rather normal pneumoencephalogram (Fitch and Pigott). The extremities of this patient were negative. Older writers (Foville) would be tempted to place such a lesion in the formatio reticularis of the brain stem. We have shown (Pike, Notkin, Coombs and Weingrow) that this is unlikely. What is left for such a case of bilateral cranial nerve involvement with epilepsy is therefore the diencephalon.

We will not linger further on this subject but proceed to present a quotation by Clark to show that all these theories cited above are beginning to give way to a physiological explanation of this phenomenon on a bioelectric basis:

"Again, Higier describes a case of periodic paralysis not consecutive to convulsions. He refers to the experiments of Sherrington (1893) and Mislawski (1898) to the effect that, if a given portion of the cerebral cortex is electrically excited, some muscles act spasmodically while others are relaxed; if flexors contract, the opposing extensors are relaxed. He therefore concludes that if the intensity of the cortical irritant is of low degree, paralysis may result instead of convulsions. This may, in fact, be demonstrated in part by experiments."

The reader is referred to the paper by Clark for further elaboration on this subject of exhaustion paraplegia, monoplegia, hemiplegia, paralysis of the abdominal muscles due to exhaustion and other phenomena which deal with the subject of neurological sequelae of an epileptic attack and their explanation. It seems most of these fall into the group of data which we showed by referring to our gastrointestinal aurae which were associated with periodic exhaustion paralysis after attacks, before we began our reference to Clark. As neurologists we might feel qualified to discuss paralysis in general, but when it becomes complicated by neurophysiologic principles it gets out of our realm. Periodic paralysis is seen in diseases under that name and in multiple sclerosis. In such conditions it is not accompanied by convulsive phenomena that lead to exhaustion of the neural or muscular elements. In status epilepticus we see exhaustion causing cardiac cessa-

tion. It is therefore not unlikely that such processes might affect viscera and even metabolism and be responsible for internal chemical changes that one epileptologist finds and another denies. From a practical standpoint such paralyses exist, but are they of focal diagnostic value? That all of the data which we presented in the above connection are related to exhaustion paralysis may be seen from the following quotation by Clark: "Occasionally there is partial loss of speech especially after right-sided seizures, often associated with right-sided weakness (as in permanent aphasia with right hemiplegia). This loss of speech may occur without weakness, especially after slight fits beginning in the face." At this point and before presenting the historical data on the 300 cases in which hemiplegias are dealt with, it is important that we impress the reader with the fact that items such as the last mentioned one, could be picked from our 5,000 cases and shown to be present in at least over 1 per cent of epileptic individuals at one or another time in their lives. Prior to direct quotation from Clark we spoke of temporary paralysis as being of diencephalic origin. The exhaustion paralysis is really not a paralysis. It is more an exhaustion, or fatigue, or loss of tone or loss of muscle strength. In this respect it resembles a myasthenic state. As such it can be conceived as diencephalic, as in myasthenia gravis. Moreover, by diencephalic we often mean thalamic. In the thalamic syndrome we often encounter flaccidity and paralysis and loss of tone. This does not have to be due to interference with the thalamostriate connections but can come from the thalamorubral and rubrodentate system. The flaccidity of the extremities in cerebellar disease is also through the same mechanism. The reader will therefore note that our title, corticodiencephalic, is a very encompassing term, and that there are many thalamic syndromes will be seen from our cases.

We were rather reticent about dragging in other subjects, that is, the subjects of postparoxysmal paralysis and postparoxysmal aphasia, and other phenomena that are interrelated and occur after a fit, but as the reader will see, we could not help ourselves, because some of these are rather common in not only epileptic seizures but also convulsions from various origins. These are therefore intimately connected with the neurogenic control of the gastrointestinal tract when it is upset, not only by the irritative processes as seen when a gastrointestinal as well as urinary aura precede the attack, but also in general convulsions. The reader will therefore realize that we were faced with a problem which involved not only the spinothalamic and pyramidal pathways that might be concerned with neurogenic control of gastrointestinal and urinary function, but with one which especially involved the corticothalamomammillary system. We have therefore the above problem in addition to the literature on the trophic fibre connections which, as we have shown by the references in Archambault and Fromm, indicates that trophic phenomena are also localized in the corticothalamomammillary and related systems. It is particularly with reference to the extensive literature shown by Archambault and Fromm that the diencephalic portion of the mammillothalamic tract was held under suspicion as containing trophic centers particularly situated around ependymal gray matter of the third ventricle, that of the aqueduct of Sylvius and other neighboring

places. With the periodicity, and the multiple combinations of the various types of so-called exhaustion paralyses, particularly of the paraplegic type, it is evident that the diencephalon seems the most likely place for the localization of such paralyses. Just as the mammillothalamic system can be expanded, because of its fibre connections in the mammillary region and in the thalamus, so can the spinothalamic system. Spiegel and others concentrated on those spinothalamic fibres (crossed and uncrossed) that carry pain impulses. From the types of gastrointestinal aurae, we can see that the spinothalamic fibres from the posterior columns, implicating the rubrothalamic through the dentorubral system, could also be included in the term "interrupted" spinothalamic. Thus sensations such as pressure vibrations and constructions in the epigastrium would be carried in the last mentioned pathways rather than in those stressed by Spiegel and other investigators to which he refers. In giving a detailed account of the anatomy of the dorsal thalamus we have therefore served all schools.

Instead of the term exhaustion paralysis it seems to us that it might be better were we to employ a different term since it is as yet uncertain as to whether fatigue, chemicals, or bioelectric processes are responsible for this type of periodic paralysis, aphasia, paraplegia, etc., etc. For this reason we propose the term of postparoxysmal periodic paralysis. In order to show that this form of paralysis is not unlikely to occur in the cases of epilepsy that have gastrointestinal and other autonomic aurae, and furthermore that various combinations of paralysis of the crossed and uncrossed type as well as of the paraplegic type exist in a rather large percentage of epileptics, we present the incidence of motor and muscle strength disturbances in 300 cases whose histories have been studied in detail.

Paralysis of the nonexhaustion type occurred not infrequently. From the above situation, from the admission records of the 300 cases which we are analyzing, one might be disappointed if one looks merely for the time paralysis in the records. If the records of the examination is analyzed for items such as disturbances in gait, deformities, spasticities, gait defects, and strabismus, one will find an appreciable amount of material. In the cases referred to, we have noticed complete paralysis occurring in three places on the left side of the body. One of the patients also shows a complete paralysis of the left side of the body. One of the patients shows a paralysis of the spinal region.

One of the patients shows a crossed paralysis involving the right arm and the left leg. Another patient shows generalized muscular rigidity. In three patients there were involvements and deformities on paralysis in the cranial nerves. No specification was made as to whether the paralysis was of the pyramidal or of the extrapyramidal variety. Some of it might have been of the reflex variety, i.e., due to hypertonia with exaggerated reflexes. There is a great deal of this type of "palsy" in institutional epileptics. It is called Little's disease and goes under the heading of various names. The relationship between the reflex type of movement and the regular orthodox type of movement we do not know. Some authorities such as R. Lorente de No call nystagmus an alternating reflex related to the scratch reflex.

Nystagmus differs little from palatal myoclonus which many authorities class under abnormal involuntary movements.

In each instance partial paralysis is associated with deformities of parts of the body. In eight patients one extremity was involved. In the seven remaining, two extremities were affected. Out of the first eight patients who presented deformities associated with paralysis of one extremity this affected the right side in four cases and the left in the rest. Those who showed disturbances on the right side showed the following characteristics. A shuffling of the right leg was present in one patient. A second patient showed an external rotation of the right leg. A third patient showed a foreshortened arch of the right foot while a fourth patient had a talipes varus of the right side. The patients who showed involvements in the left lower extremity were characterized by the following defects. One patient walked with his body leaning on the ball on his left foot. A third patient suffered from a slight spasticity of the left lower extremity, while a fourth individual had a contracture of his left leg. Three patients who suffered from involvements of both lower extremities were described in the following ways. One patient was said to have a spastic gait. A second patient was described as creeping instead of walking, because of deformed feet, while a third patient was said to have a shuffling gait. The last four cases that were said to be paraplegic were described in the following way. One patient was said to walk with his feet spread widely apart. A second patient was said to have a bilateral congenital paraplegia. A third patient was said to suffer from a flaccidity of the lower limbs while a fourth one, was said to have an unsteady gait and described as dragging of his feet in walking.

Aside from the above data, various types of deformities were present some of them affecting the spinal column in eleven patients. Strabismus was present in three patients. Besides actual paralyses there were reflex disturbances which pointed to a possible existence of some form of paralysis or change in muscle tone. Muskens has developed the intimate relationship between reflex reactions and epileptic myoclonic responses. Stanley Cobb illustrated the bioelectric mechanisms operating in these. The clonic responses seen in such seemingly simple phenomena as ankle clonus and patellar clonus still lack fundamental physiological facts. R. Lorente de No and Barany have however contributed much by their work upon nystagmus. From the experiments connected with this one sees in the reflex mechanism an active component and a passive one. All the neural structures between the former and the latter, consisting of the internuncials, seem to serve as modifiers of the relationships between the first and the second components. Thus the tonic phase consists of, as shown by Muskens and Cobb, a prongation of the active phase. Muscle strength might also be placed in this category. To what extent gastrointestinal muscle tone enters into the above is a problem for future workers. Reflex clonus was present in four patients. The Babinski, Oppenheim, Gordon and Chaddock reflexes were positive in nine patients individually. The abdominal reflexes were affected in one case, while in six other patients the tendon reflexes were variously involved. Although no mention was made of the Hoffman and Kernig signs

there is no doubt that some of these signs as well as the sign of Romberg and others were positive. We have observed the groups of scratch reflex reactions in epileptics but have reserved comment for the reason that we are at present unable to express opinions with regard to such metaphysical problems as relate reflex to motion. We feel that this has been a little overdone in epilepsy. Muskens has written a whole volume on myoclonic shock reflexes and has concentrated upon the mechanism of "reflex after discharge". Yet few have referred to him. Even though Musken's original ideas were obtained from his experience with the vestibular pathways and with the closed circuits connected with reticular functioning we see very little in the literature emanating from workers with the above structures who might comment upon myoclonic shock reflexes. If this has been done, it hasn't been circulated enough to attract our attention.

A number of authorities have written on the appearance of abnormal reflexes which occur after a fit. Such reflexes seem to be present in individuals who do not show them independent of fits. Whether these might be explained on the basis of exhaustion or not is an interesting problem. It stands to reason that a pupillary reaction might become fatigued due to repeated contraction during a fit. Although many outstanding authorities such as Gowers and Oppenheim and others have written on abnormal reflexes in epilepsy, no one seems to have concentrated upon it in relation to paralysis of all types which occur in epileptic patients. If this were done the problem of exhaustion paralyses might be solved.

We introduced the data upon the motor disturbances of the gastrointestinal tract by presenting the literature on the above subject in epilepsy up to the year 1929, as analyzed by Lennox and Cobb. The analysis of material dealing with disturbances in motion and tone of the gastrointestinal musculature in epilepsy was rather extensive. Although many authorities were cited in that particular analysis, it was shown that very little attention was paid to the tracts within the brain and the spinal cord which carried impulses that mediated the above functions in the normal individual. Not only was the normal insufficiently stressed but the lesions within the brain of epileptics that might produce abnormalities in gastrointestinal functions were completely overlooked. The excuse for the complete omission of the above was emphasized in the subsequent portions of this paper. The reasons were given in the analysis of the literature by Donal Sheehan and Watts and Fulton respectively. It was shown by the last mentioned authors that even in the nonepileptic the cortical nerve center and tract background controlling gastrointestinal functions were far from definitely established. Although we have repeatedly referred to the substantia reticularis, to the medial and posterior longitudinal fasciculus, to the tract of Darkschewitsch and the related structures as connected to the mammillothalamic system, and to the extrapyramidal pathways by collateral fibres, we hesitate to repeat the same in relation to the Watts and Fulton hypothesis that gastrointestinal function operates particularly through the last mentioned channels. It is, however, not unlikely that Fulton especially, might have been responsible for such leanings towards the internuncial and rhythmic reflex structures. If the last

mentioned statement be true and the neurogenic gastrointestinal central centers operate along such mechanisms, then the problems of cortical reciprocal innervation which faced them, could be explained on the basis of passive inhibition as proposed by R. Lorente de No for the vestibuloocular reflex arc (1933). It remains therefore for us to be among the first to present especially chosen and worked-up clinical material in epilepsy, which would show that, in patients suffering from gastrointestinal aurae preceding the convulsion, there are definite brain diseases which are responsible for the former as well as the latter. We intend to embark upon the above mentioned project in the form of group syndromes into which we will segregate special cases which suffer from aurae of abdominal pain, vomiting, nausea, as well as other gastrointestinal complaints. We will not only give the number of cases in each group, but will also analyze a number of individual case records, and whereever data upon pneumoencephalograms and subdural air studies are available we shall also include them. The effects of carotid sinus compression and special seizure records will be added when available. Before entering upon the first group syndrome which deals with abdominal pain, we will present two outstanding cases in which the clinical as well as x-ray findings of gastrointestinal disturbances are described in detail. We are doing so mainly because we wish to impress the reader with the fact that an aura referring to the gastrointestinal tract, no matter how insignificant, might be accompanied by contractions or dilatations of the stomach and of the small as well as the large intestines. Not only might an aura, particularly one which is associated with nausea or vomiting, indicate dysfunction of the above muscular structures but of the gastrointestinal sphincters and of mucosa as well.

(To be continued)

CHAPTER ACTIVITIES

Argentina Chapter

At a meeting of the Argentina Chapter of the National Gastroenterological Association the following officers were elected for the year 1950-1951: President, Dr. Oscar Cames; Vice-President, Dr. Juan Cortes; Secretary-General, Dr. Luciano M. Torres; Treasurer, Dr. Oscar Pardo; Secretary, Dr. Daniel Gorni; Secretary in charge of publications, Dr. Norberto Baravalle; Members of the Executive Board, Dr. Ricardo Delgado, Dr. Antonio Manzoni, Dr. Atilio Rossi and Dr. Jose Aused Callau.

New Jersey Chapter

The New Jersey Chapter of the National Gastroenterological Association held a business meeting at the Berkeley-Carteret Hotel in Asbury Park, N. J. on Sunday, 11 June 1950.

The business session was preceded by a scientific program at which Dr. Edward Berk of Temple University spoke on the subject of "Pancreatitis, Acute and Chronic".

The following officers were elected for the year 1950-1951: President, Dr. Sidney Rosenthal, Newark, N. J.; Vice President, Dr. Andrew J. V. Klein, Short Hills, N. J.; Secretary-Treasurer, Dr. Benjamin J. Macchia, Jersey City, N. J.; Executive Council to consist of Drs. Samuel H. Rubin, Asbury Park, N. J.; Arthur J. Statman, Newark, N. J.; S. William Kalb, Newark, N. J.; Leonard Troast, Jersey City, N. J.; Representatives to the National Council, Drs. Earl J. Halligan, Jersey City, N. J. and Hyman I. Goldstein, Camden, N. J.

Following the meetings of the New Jersey Gastroenterological Society a testimonial dinner was given to Dr. Hyman I. Goldstein who was presented with a certificate from the New Jersey Chapter naming him the "Gastroenterologist of the Year" of the New Jersey Chapter.

New York Chapter

The Annual Meeting of the New York Chapter of the National Gastroenterological Association was held at the home of Dr. Samuel Weiss, New York City on Monday evening, 15 May 1950.

The following officers were reelected for the 1950-51 season: Drs. William C. Jacobson, President; Isidor L. Ritter, Vice-President; Harry Barowsky, Secretary and Elihu Katz, Treasurer.

By unanimous vote of those present, the Constitution of the Chapter was amended to establish two new offices, those of Second Vice-President and Recording Secretary of the Chapter. Dr. Frederic W. Bancroft was elected Second Vice-President and Dr. A. X. Rossien, Recording Secretary.

The next regularly scheduled meeting of the New York Chapter will be held at the New York Academy of Medicine in November.

Further details will be published as they become available.

NEWS NOTES

Fifteenth Annual Convention

The program for our Fifteenth Annual Convention is bound into this issue of The Review of Gastroenterology and will not be mailed separately to the members of the Association.

Additional copies are available from the headquarters office, 1819 Broadway, New York 23, N. Y. The program will also be available at the registration desk on the Convention floor.

As a result of the decision of the Executive Board following the Fourteenth Annual Convention, the program for Monday afternoon, 9 October 1950, has been limited to two papers in order to allow sufficient time for the business meeting and the Convocation.

To compensate for this an additional session has been scheduled for Wednesday evening, 11 October 1950, for the presentation of additional excellent papers.

Registration

All those attending the Convention are requested to register and receive their identification badges. Ladies are also invited to register. None will be admitted to the exhibits or the sessions without a badge.

Annual Meeting of the National Council

The National Council of the National Gastroenterological Association will hold its Annual Meeting at the Hotel Statler in New York City on Sunday afternoon, 8 October 1950, at 4:00 P.M.

Delegates of chapters will present reports of their chapter's activities for the past year.

Annual Meeting of the National Executive Board

The Annual Meeting of the National Executive Board will be held at the Hotel Statler in New York City on Sunday afternoon, 8 October 1950, at 5:00 P.M.

Annual Meeting of the National Gastroenterological Association

The Annual Meeting of the National Gastroenterological Association will be held at the Hotel Statler in New York City at 4:00 p.m. on Monday, 9 October 1950, the first day of the Fifteenth Annual Convention.

Members of the Association are invited to attend and participate in the business meeting.

The proposed revision of the Constitution will be acted upon at that time.

Convocation Ceremony

The Convocation Ceremony, at which certificates of affiliation will be presented in person to newly elected Members, Associate Fellows, Fellows and those advanced in the various categories during the year, will follow the Annual Meeting of the Association at 5:00 p.m. on Monday afternoon, 9 October 1950, at the Hotel Statler in New York City.

This year, those participating in the Convocation Ceremony will wear academic costume, and an interesting program has been planned.

Members, their families, guests and friends are invited to attend.

President's Annual Reception

The President's Annual Reception, again sponsored by Winthrop-Stearns, Inc., will be held immediately following the Convocation Ceremony on Monday evening, 9 October 1950, at 6:30 p.m. Members of the Association, their friends, guests, as well as those present with the technical exhibits are cordially invited to attend. Admission cards may be secured at the time of registration at the registration desk.

Annual Banquet

The Annual Banquet of the National Gastroenterological Association will be held at the Hotel Statler in New York City on Tuesday evening, 10 October 1950 at 7:00 p.m., to be preceded by cocktails.

Course in Postgraduate Gastroenterology

Immediately following our Fifteenth Annual Convention, the National Gastroenterological Association is conducting a Course in Postgraduate Gastroenterology on 12, 13, 14 October 1950, at the Hotel Statler in New York City.

The Course this year again will be under the personal direction of Dr. Owen H. Wangensteen, Professor and Chairman of the Department of Surgery, University of Minnesota Medical School, who also will be surgical coordinator. The Associate Director of the Course will be Dr. I. Snapper of The Mt. Sinai Hospital, who will be the medical coordinator.

Participating in the Course will be a distinguished faculty chosen from the medical schools in and around New York City.

Enrollment for the Course is still open and admission will be limited to those holding matriculation cards indicating that they have paid the $35.00 fee for the three days.

Applications and further information may be obtained by writing to the National Gastroenterological Association, 1819 Broadway, New York 23, N. Y.

Regional Meeting

Representatives of the west coast and midwest chapters of the National Gastroenterological Association met with representatives of the National Executive Board on 26 June 1950, at the office of Dr. Felix Cunha in San Francisco, Calif.

Those attending the meeting included Dr. George P. Gray, President of the San Francisco Chapter, Dr. Arthur Kirchner, President of the Los Angeles Chapter and Dr. L. Dale Huffman of the Los Angeles Chapter, Dr. John E. Cox, President of the Memphis Chapter, Dr. L. Dale Creek, representing the Santa Barbara Chapter, Dr. Felix Cunha, Vice-President of the National Gastroenterological Association and representing the San Francisco Chapter, Dr. William W. Lermann, Vice-President of the National Gastroenterological Association and Mr. Daniel Weiss, Executive Officer.

The group discussed the advisability of having a regional meeting to include the west coast and midwest chapters and tentatively set the dates for sometime in April.

Further details concerning this meeting will be published as they become available.

Executive Board Meetings

A regular meeting of the Executive Board of the National Gastroenterological Association was held on Sunday morning, 19 March 1950 in New York City.

Routine correspondence and administrative matters were properly disposed of.

The following new members were elected to the National Gastroenterological Association: Drs. Francis M. Burke, Natick, Mass., Member; Robert Vincent Condon, Brookline, Mass., Associate Fellow; Edward A. Cooney, Boston, Mass., Fellow; Gerald Lewis Haidak, Pittsfield, Mass., Member; Melvin P. Osborne, Brookline, Mass., Member; Anthony S. Ripa, East Boston, Mass., Member; George Robbins, Boston, Mass., Member; Isadore Schwartz, Quincy, Mass., Member and William E. Fradkin, Brooklyn, N. Y., Member.

Upon presentation of the necessary qualifications the following were advanced as indicated: Drs. Arden S. Turner, Philadelphia, Pa., Associate Fellow; Stanley L. Moleski, Grand Rapids, Mich., Associate Fellow; Kenneth C. Peacock, New York, N. Y., Fellow; C. H. Benage, Pittsburgh, Kansas, Associate Fellow; David B. Monheit, Brooklyn, N. Y., Associate Fellow; Earl J. Halligan, Jersey City, N. J., Fellow; G. Irving Levine, Jersey City, N. J., Associate Fellow; Thomas H. Cuddy, Winnipeg, Manitoba, Canada, Fellow; Stanley Sidenberg, Cleveland, Ohio, Associate Fellow; Abraham J. Brenner, Brooklyn, N. Y., Associate Fellow; G. N. Fluegal, Wilkes-Barre, Pa., Associate Fellow; Milton Fastenberg, New York, N. Y., Associate Fellow; Francisco D'Imperio, Camden, N. J., Associate Fellow; Jacob A. Riese, West New York, N. J., Associate Fellow; Charles M. Rothburd, New York, N. Y., Associate Fellow; Maurice Bruce Bangel, Brooklyn, N. Y., Associate Fellow; J. L. Chereskin, Springfield, Mass., Fellow; William F. Herzig, New York, N. Y., Associate Fellow; Norman A. Samuels, New York, N. Y., Associate Fellow;

Program

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

FIFTEENTH ANNUAL CONVENTION
9, 10, 11 OCTOBER 1950
and
COURSE IN GASTROINTESTINAL SURGERY
12, 13, 14 OCTOBER 1950

HOTEL STATLER
Seventh Avenue and 33rd Street
New York, N. Y.

Members of the medical profession
are cordially invited to attend the convention sessions.

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REGISTRATION—All members and guests should register. Identification badges for admittance to meetings will be given to those who register. These should be worn at all times during the session. Registration will take place at the registration desk on the convention floor.

LADIES REGISTRATION—At the registration desk on the Convention Floor. Registration facilities will be open at 8:30 each morning. Information concerning the various activities and events will be available there.

MEETINGS are held on Eastern Standard Time and will begin promptly at the time specified.

COURSE IN POSTGRADUATE GASTROENTEROLOGY—Admittance only upon presentation of official matriculation card.

SCIENTIFIC EXHIBITS—Will be in the Exhibit Hall and will be open daily, Monday to Thursday.

TECHNICAL EXHIBITS under the direction of Mr. Steven K. Harlitz, Exhibit Manager, will be open Monday to Thursday.

Those attending the Convention are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with the many new products and new equipment on display.

VISIT THE EXHIBITS

Program

FIFTEENTH ANNUAL CONVENTION
NATIONAL GASTROENTEROLOGICAL ASSOCIATION

SCIENTIFIC SESSIONS

9, 10, 11 OCTOBER 1950

HOTEL STATLER

Seventh Avenue and 33rd Street

New York, N. Y.

Members of our Association and subscribers to THE REVIEW OF GASTROENTEROLOGY, whether present or not at the time of the reading of a paper are always looking forward to its being published, so that they may read it carefully. With that in mind the essayists have been asked to submit their papers for publication.

FIRST SESSION

MONDAY MORNING, 9 OCTOBER 1950

HORACE W. SOPER, M.D., President, National Gastroenterological Association, Presiding.

9:00 A.M.

1. **"Some Unusual Forms of Perforation Complicating Benign Peptic Ulcer and Gastric Carcinoma".**

Speaker

MAURICE FELDMAN, M.D., Baltimore, Md., Assistant Professor of Gastroenterology, University of Maryland School of Medicine (By invitation).

9:30 A.M.

Discussion to be opened by:

FRANK J. BORRELLI, M.D., Tuckahoe, N. Y.

9:50 A.M.

2. **"Jejunum Histopathological Studies Through Surgical Biopsy in a Case of Active Tropical Sprue".**

Speakers

FERNANDO MILANES, M.D., Havana, Cuba, Professor of General Pathology, School of Medicine, University of Havana; LEON BLANCO P., M.D., Havana, Cuba, Professor of Pathology, School of Medicine, University of Havana and A. CAUSA, M.D., Havana, Cuba, Instructor of General Pathology, School of Medicine, University of Havana.

10:20 A.M.

General Discussion

10:40 A.M. Recess to visit the Commercial and Technical Exhibits.

VISIT THE EXHIBITS

11:00 A.M.

3. "Six Hundred Cases of Acute Perforated Peptic Ulcer".

Speakers

JOHN B. O'DONOGHUE, M.D., F.A.C.S., F.I.C.S., Chicago, Ill., Clinical Professor of Surgery, Stritch School of Medicine, Loyola University and MAURICE B. JACOBS, M.D., F.I.C.S., F.A.C.S., Chicago, Ill., Associate Attending Surgeon, Cook County Hospital, Staff Surgeon, University Hospital (By invitation).

11:30 A.M.

General Discussion

11:50 A.M.

LUNCHEON AND ROUND-TABLE DISCUSSION.

Subject

"One Hundred Years of Gastroenterology (1850-1950)".

Speaker

HYMAN I. GOLDSTEIN, M.D., Camden, N. J., Historian, National Gastroenterological Association.

SECOND SESSION

MONDAY AFTERNOON, 9 OCTOBER 1950

WILLIAM W. LERMANN, Vice-President, National Gastroenterological Association, Presiding.

2:00 P.M.

4. "The Prevention of Some Postgastrectomy Difficulties by a New Gastrectomy Technic. (Pantaloons Anastomosis)".

Speaker

MOSES E. STEINBERG, B.S., M.S., M.D., Portland, Ore., Attending Staff, Emanuel Hospital, Portland, Oregon, Associate in Physiological Research, University of Oregon Medical School.

2:30 P.M.

Discussion to be opened by:

B. O. C. PRIBRAM, M.D., New York, N. Y.

2:50 P.M. Recess to visit the Commercial and Technical Exhibits.

3:10 P.M.

5. "Bleeding Peptic Ulcer".

Speaker

EDWARD T. LEWISON, B.S., M.D., Baltimore, Md., Surgeon, Johns Hopkins Hospital, Instructor in Surgery, Johns Hopkins University School of Medicine (By invitation).

3:40 P.M.

Discussion to be opened by:

LAURENCE MISCALL, M.D., New York, N. Y. (By invitation).

4:00 P.M.

ANNUAL MEETING OF THE ASSOCIATION—GENERAL ASSEMBLY.

5:00 P.M.

CONVOCATION: Presentation of Certificates.

See Special Program.

6:30 P.M.

PRESIDENT'S ANNUAL RECEPTION (Admission by card only).

THIRD SESSION

TUESDAY MORNING, 10 OCTOBER 1950

CHARLES W. MCCLURE, M.D., Vice-President, National Gastroenterological Association, Presiding.

9:00 A.M.

6. "Surgical Treatment of Congenital Types of Diaphragmatic Hernia".

Speaker

STUART W. HARRINGTON, M.D., M.S., Rochester, Minn., Professor of Surgery, Mayo Foundation Graduate School, University of Minnesota, The Mayo Clinic, Rochester, Minn. (By invitation).

9:30 A.M.

Discussion to be opened by:

ROY UPHAM, M.D., New York, N. Y.

9:50 A.M.

7. "The Increased Incidence of Coronary Thrombosis in Peptic Ulcer".

Speaker

LESTER M. MORRISON, M.D., F.A.C.P., Los Angeles, Calif., Los Angeles County General Hospital, The Cedars of Lebanon Hospital and the College of Medical Evangelists.

10:20 A.M.

Discussion to be opened by:

DAVID ADLERSBERG, M.D., New York, N. Y.

10:40 A.M. Recess to visit the Commercial and Technical Exhibits.

11:00 A.M.

8. "The Choice of Treatment in Idiopathic Dilatation of the Esophagus".

Speakers

W. J. MERLE SCOTT, M.D., Rochester, N. Y., Associate Professor of Surgery, University of Rochester Medical School, Associate Surgeon, Strong Memorial Hospital and GEORGE EMERSON, M.D., Rochester, N. Y. (By invitation).

11:30 A.M.

Discussion to be opened by:

DAVID J. BARRY, M.D., New York, N. Y. (By invitation).

VISIT THE EXHIBITS

FOURTH SESSION

TUESDAY AFTERNOON, 10 OCTOBER 1950

HARRY M. EBERHARD, M.D., Vice-President, National Gastroenterological Association, Presiding.

1:00 P.M.

9. "Achlorhydria and Hypochlorhydria Associated with Chronic Urticaria".

Speakers

WILLIAM B. RAWLS, B.S., M.D., New York, N. Y., Attending Physician, Polyclinic Postgraduate Medical School and Hospital and V. CHARLES ANCONA, M.D., New York, N. Y., Assistant Attending Physician, St. Clare's Hospital.

1:30 P.M.

Discussion to be opened by:

SAMUEL BERGER, M.D., Cleveland, Ohio.

1:50 P.M.

10. "Gastrointestinal Function Following Radical Pancreaticoduodenectomy".

Speakers

WILLIAM OSTROW, M.D., Brooklyn, N. Y., Chief of Department of Gastroenterology, Coney Island Hospital, Visiting Gastroenterologist, Coney Island Hospital, Brooklyn, N. Y., and CHARLES B. RIPSTEIN, M.D., F.R.C.S., Associate Professor of Surgery, State University Medical Center, Director of Surgical Services, Maimonides Hospital, Brooklyn, N. Y.

2:20 P.M.

General Discussion

2:40 P.M. Recess to visit the Commercial and Technical Exhibits.

3:00 P.M.

11. "Pancreatic Resection for Benign Diseases of the Pancreas".

Speaker

RICHARD B. CATTELL, M.D., Boston, Mass., Surgeon-in-Chief, New England Deaconess Hospital, Boston, Surgeon, New England Baptist Hospital, Boston, Mass.

3:30 P.M.

General Discussion

7:00 P.M.

ANNUAL BANQUET—HOTEL STATLER, New York, N. Y.

VISIT THE EXHIBITS

FIFTH SESSION

WEDNESDAY MORNING, 11 OCTOBER 1950

WILLIAM C. JACOBSON, M.D., President, New York Chapter, National Gastroenterological Association, Presiding.

9:00 A.M.

12. "Results of Routine Sigmoidoscopy".

Speaker

VINCENT T. YOUNG, M.D., C.M., Washington, D. C., Instructor in Proctology, Georgetown Medical School, Proctologist, Yater Clinic (By invitation).

9:30 A.M.

Discussion to be opened by:

EMIL GRANET, M.D., New York, N. Y.

9:50 A.M.

13. "The Diagnosis and Treatment of Colonic Stasis".

Speaker

HARRY BAROWSKY, B.S., M.D., New York, N. Y., Assistant Clinical Professor, Department of Medicine, New York Medical College.

10:20 A.M.

Discussion to be opened by:

WILLIAM W. LERMANN, M.D., Pittsburgh, Pa.

10:40 A.M. Recess to visit the Commercial and Technical Exhibits.

11:00 A.M.

14. "The Evolution of Surgical Supervision in the Management of Advanced Idiopathic Ulcerative Colitis".

Speakers

HARRY E. BACON, M.D., F.A.C.S., F.I.C.S., Philadelphia, Pa., Professor and Head of the Department of Proctology, Temple University School and Hospital, Head of Department, St. Mary's Hospital and HOWARD D. TRIMPI, A.B., M.S., M.D., Philadelphia, Pa., Associate in Proctology, Temple University Hospital.

11:30 A.M.

Discussion to be opened by:

SCUDDER WINSLOW, M.D., New York, N. Y. (By invitation).

SIXTH SESSION

WEDNESDAY AFTERNOON, 11 OCTOBER 1950.

C. J. TIDMARSH, M.D., President, National Gastroenterological Association, Presiding.

2:00 P.M.

15. "Gastroscopic Biopsy; Correlation of Clinical Findings and Tissue Biopsy of the Stomach".

Speakers

PAUL L. SHALLENBERGER, M.S., M.D., Sayre, Pa., Chief, Section of Gastroenterology, Guthrie Clinic and Robert Packer Hospital; CHARLES H. DE-

WAN, B.S., M.D., Chief, Section of Pathology, Guthrie Clinic and Robert Packer Hospital; CLAYTON B. WEED, A.B., M.D., Resident in Medicine, Guthrie Clinic and Robert Packer Hospital and JOHN C. REGANIS, B.S., M.D., Sayre, Pa., Resident in Pathology, Guthrie Clinic and Robert Packer Hospital (By invitation).

2:30 P.M.

Discussion to be opened by:

JEAN LESAGE, M.D., Montreal, Canada.

2:50 P.M.

16. "Clinical X-ray Conference on Localized Constricting Lesions of the Colon".

Speakers

LYNN A. FERGUSON, B.S., M.D., Grand Rapids, Mich., Chief of Surgical Division, Ferguson-Droste-Ferguson Rectal Clinic and Hospital, Consulting Surgeon, Division of Proctology and Colon Surgery; BENJAMIN R. VAN ZWALENBURG, B.S., M.D., Grand Rapids, Mich., Consulting Radiologist, Ferguson-Droste-Ferguson Rectal Clinic and Hospital, Radiologist, Holland City Hospital and EDWARD F. DUCEY, B.S., M.D., F.A.C.P., Grand Rapids, Mich., Pathologist, Ferguson-Droste-Ferguson Rectal Clinic and Hospital, Pathologist, St. Mary's Hospital.

3:20 P.M.

Discussion to be opened by:

A. J. TILLINGHAST, M.D., New York, N. Y. (By invitation).

3:40 P.M. Recess to visit the Commercial and Technical Exhibits.

4:00 P.M.

17. "Surgery in Massive Hemorrhage of the Upper Gastrointestinal Tract".

Speakers

EARL J. HALLIGAN, M.D., F.A.C.S., F.I.C.S., Jersey City, N. J., Chief Surgeon, St. Francis' Hospital, Consulting Surgeon, Jersey City Medical Center, Attending Surgeon, Margaret Hague Maternity Hospital, Consulting Surgeon, St. Mary's Hospital, Hoboken, N. J., Consulting Surgeon, Holy Name Hospital, Teaneck, N. J.; LOUIS L. PERKEL, M.D., F.A.C.P., Jersey City, N. J., Attending Physician and Chief of the Department of Gastroenterology, Jersey City Medical Center, Consultant Gastroenterologist to Margaret Hague Maternity Hospital, B. B. Pollak Hospital for Chest Diseases and North Hudson Hospital, Weehawken, N. J.; J. KENNETH CATLAW, M.D., A.I.C.S., Jersey City, N. J., Visiting Surgeon, St. Francis' Hospital, Assistant Visiting Surgeon, Jersey City Medical Center and LEONARD TROAST, M.D., Jersey City, N. J., Assistant Attending Gastroenterologist, Jersey City Medical Center.

4:20 P.M.

Discussion to be opened by:

FREDERIC W. BANCROFT, M.D., New York, N. Y.

SEVENTH SESSION

WEDNESDAY EVENING, 11 OCTOBER 1950.

ANTHONY BASSLER, M.D., F.A.C.P., LL.D., Honorary President, National Gastroenterological Association; ROY UPHAM, M.D., F.A.C.S., Secretary-General, National Gastroenterological Association and SIGURD W. JOHNSEN, M.D., Secretary, National Gastroenterological Association, Co-Chairmen.

8:00 P.M.

18. "Treatment of Chronic Nonspecific Ulcerative Colitis with Aureomycin".

Speakers

JEROME A. MARKS, A.B., M.D., New York, N. Y., Assistant Professor of Clinical Medicine, New York University-Bellevue Medical Center; LOUIS T. WRIGHT, A.B., M.D., F.A.C.S., Director of Surgery, Harlem Hospital and SELIG STRAX, B.S., M.D., F.A.C.S., New York, N. Y., Associate Visiting Surgeon, Harlem Hospital, Senior Clinical Assistant Visiting Surgeon, The Mt. Sinai Hospital.

8:15 P.M.

19. "The Postdysentery Syndrome".

Speakers

SIDNEY M. FIERST, B.S., M.D., M.S., Brooklyn, N. Y., Associate in Medicine, State University College of Medicine at New York, Assistant Attending Physician, Maimonides Hospital, Long Island College Hospital and Kings County Hospital, Chief of Gastrointestinal Clinic, Maimonides Hospital, Assistant Chief, Gastric Clinic, Long Island College Hospital, Gastroenterologist, Brooklyn Regional Office, Veterans Administration and ABRAHAM WERNER, B.S., M.D., Brooklyn, N. Y., Chief, Internal Medicine Section, Brooklyn Regional Office, Veterans Administration.

8:30 P.M.

20. "Is Surgery Necessary for the Silent Gallstone?"

Speaker

JACOB A. RIESE, A.B., M.D., Jersey City, N. J., Associate Attending Gastroenterologist, Jersey City Medical Center.

8:45 P.M.

21. "Small Bowel Physiology: Recent Experimental Observations".

Speaker

BERNARD J. FICARRA, A.B., Sc.B., M.D., Brooklyn, N. Y., Associate Visiting Surgeon, Brooklyn Cancer Institute, St. Peter's Hospital, Hospital of Holy Family, Assistant Visiting Surgeon, Kings County Hospital.

9:00 P.M.

22. "Melanosis Coli".

Speakers

LOUIS L. PERKEL, B.S., M.D., F.A.C.P., Jersey City, N. J., Attending Gastroenterologist, Jersey City Medical Center and LEONARD TROAST, B.S., M.D., Jersey City, N. J., Assistant Attending Physician, Jersey City Medical Center.

VISIT THE EXHIBITS

9:15 P.M.

23. "The Use of Aureomycin in Amebiasis".

Speaker

JOHN E. COX, M.D., Memphis, Tenn.

9:30 P.M.

24. "Jaundice as a Complication of Gastric Resection".

Speakers

JOSEPH TARTAKOFF, M.D., Boston, Mass., Assistant Professor of Surgery, Tufts College Medical School, Surgical Staff, Boston City Hospital and JAMES M. MCNULTY, M.D., Boston, Mass., Former Resident Surgeon, Boston City Hospital.

9:45 P.M.

25. "Sympathetic Nerve Block for Preservation of Strangulated Intestines".

Speaker

HELMUTH NATHAN, M.D., F.I.C.S., New York, N. Y., Associate Visiting Surgeon, Sydenham Hospital, Senior Clinical Assistant, The Mt. Sinai Hospital.

10:00 P.M.

26. "Management of Upper Gastrointestinal Hemorrhage".

Speaker

NATHAN W. CHAIKIN, B.S., M.D., F.A.C.P., New York, N. Y., Assistant Professor, Clinical Medicine, New York Medical College, Flower and Fifth Avenue Hospitals, Attending Physician, Metropolitan Hospital, Chief, Gastrointestinal Clinic, Flower, Fifth Avenue and Metropolitan Hospitals, Assistant Attending Physician, Flower and Fifth Avenue Hospitals.

10:15 P.M.

27. "Prolapse of the Gastric Mucosa".

Speakers

EMANUEL M. RAPPAPORT, M.D., Jamaica, N. Y., Associate Visiting Gastroenterologist and Physician in Charge, Gastrointestinal Outpatient Department, Queens General Hospital, Associate Gastroenterologist, Triboro Hospital; EUGENE O. RAPPAPORT, M.D., Jamaica, N Y., Assistant Visiting Physician, Jamaica Hospital, Assistant Visiting Gastroenterologist, Triboro Hospital and ARNOLD STANTON, M.D., Jamaica, N. Y., Associate Physician, Jamaica Hospital, Clinical Assistant, Gastrointestinal Outpatient Department, Queens General Hospital.

COURSE IN POSTGRADUATE GASTROENTEROLOGY

OF THE

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

12, 13, 14 OCTOBER 1950

HOTEL STATLER

Seventh Avenue and 33rd Street

New York, N. Y.

DIRECTOR AND SURGICAL COORDINATOR

OWEN H. WANGENSTEEN, B.A., M.D., Ph.D., Minneapolis, Minn.

ASSOCIATE DIRECTOR AND MEDICAL COORDINATOR

I. SNAPPER, M.D., Ph.D., New York, N. Y.

OFFICERS OF INSTRUCTION

ALBERT F. R. ANDRESEN, M.D., Professor of Clinical Medicine, State University, New York State University Medical Center in New York City, College of Medicine; Attending Physician, Long Island College Hospital.

HARRY BAROWSKY, B.S., M.D., Assistant Clinical Professor, Department of Medicine, New York Medical College, Flower and Fifth Avenue Hospitals.

LEONIDAS H. BERRY, M.S., M.D., Professor of Gastroenterology and Gastroscopy, Cook County Graduate School of Medicine; Senior Attending Physician and Chairman, Department of Medicine, Provident Hospital, Chicago, Ill.

ALEXANDER BRUNSCHWIG, B.S., M.S., M.D., Professor of Clinical Surgery, Cornell University Medical College; Attending Surgeon, Memorial Hospital.

JACOB BUCKSTEIN, B.S., M.D., Assistant Professor of Clinical Medicine, Cornell University Medical College; Attending Gastroenterologist, Beth David Hospital; Visiting Roentgenologist, (Alimentary Tract Division) Bellevue Hospital.

HENRY W. CAVE, M.D., F.A.C.S., Clinical Professor of Surgery, Columbia University, College of Physicians and Surgeons; Chief, First Surgical Division and Attending Surgeon, Roosevelt Hospital.

RALPH COLP, A.B., M.D., Clinical Professor of Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, The Mt. Sinai Hospital.

BURRILL B. CROHN, M.D., Consultant in Gastroenterology, Columbia University, College of Physicians and Surgeons, Postgraduate Teaching; Consultant in Gastroenterology, The Mt. Sinai Hospital.

ARMISTEAD C. CRUMP, M.S., M.D., Formerly Associate Attending Physician in Medicine, Presbyterian Hospital.

SIDNEY FARBER, M.D., Professor of Pathology, Harvard Medical College; Pathologist in Chief, Children's Hospital, Boston, Mass.

HARRY D. FEIN, B.S., M.D., F.A.C.P., Assistant Professor of Clinical Medicine, New York University College of Medicine; Visiting Staff, Lenox Hill and Bellevue Hospitals; Attending Physician, Veterans Hospital.

FRANK GLENN, M.D., Lewis Atterbury Stimson Professor of Surgery, Cornell University Medical College; Surgeon in Chief, The New York Hospital.

J. WILLIAM HINTON, M.D., Chairman, Department of Surgery, Postgraduate Medical School of New York University; Director of Surgery, University Hospital; Director of Surgery, Fourth Surgical Division, Bellevue Hospital.

FRANKLIN HOLLANDER, Ph.D., Columbia University Faculty of Medicine; The Mt. Sinai Hospital.

J. STAUFFER LEHMAN, B.S., M.D., F.A.C.R., Professor of Radiology, Hahnemann Medical College and Hospital, Philadelphia, Pa.

FRANZ J. LUST, M.D., Chief, Gastrointestinal Clinic, Gouverneur Hospital.

THOMAS H. McGAVACK, B.A., M.D., Professor of Clinical Medicine, Director of Medical Research, New York Medical College, Metropolitan Hospital.

VISIT THE EXHIBITS

RUDOLPH NISSEN, M.D., Assistant Professor of Clinical Surgery, State University, New York State University Medical Center in New York City, College of Medicine; Attending Surgeon, Brooklyn Jewish Hospital.

GEORGE T. PACK, B.S., M.D., LL.D., Associate Professor of Clinical Surgery, Cornell University Medical College; Clinical Professor of Surgery, New York Medical College; Attending Surgeon, Memorial Cancer Center.

WILLIAM BARCLAY PARSONS, A.B., M.D., Professor of Clinical Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, Presbyterian Hospital.

JOHN L. POOL, M.D., F.A.C.S., Assistant Professor of Clinical Surgery, Cornell University Medical College; Associate Attending Surgeon, Thoracic Service, Memorial Hospital; Assistant Attending Surgeon, Lincoln Hospital, Presbyterian Hospital.

EMANUEL M. RAPPAPORT, M.D., Associate Visiting Gastroenterologist and Physician in Charge, Gastrointestinal Outpatient Department, Queens General Hospital; Associate Gastroenterologist, Triboro Hospital.

I. S. RAVDIN, M.D., John Rhea Barton Professor of Surgery, University of Pennsylvania Medical School; Director, Harrison Department of Surgical Research, University of Pennsylvania School of Medicine; Surgeon in Chief, Hospital of the University of Pennsylvania.

MARTIN E. REHFUSS, M.D., F.A.C.P., Professor of Clinical Medicine and in Charge of Therapeutics, Jefferson Hospital and Jefferson Medical College.

JOHN W. RICHTER, B.S., D.D.S., University Hospital.

LOUIS M. ROUSSELOT, A.B., M.D., M.S., Sc.D., Clinical Professor of Surgery, New York University School of Medicine; Director of Surgery, St. Vincent's Hospital.

HARRY SHAY, M.D., Professor of Clinical Medicine and Director, Samuel S. Fels Research Institute, Temple University School of Medicine.

I. SNAPPER, M.D., Ph.D., Clinical Professor of Medicine, Columbia University College of Physicians and Surgeons; Physician and Director of Medical Education, The Mt. Sinai Hospital.

H. M. VARS, Ph.D., Associate Professor of Biochemistry in Surgical Research, University of Pennsylvania Medical School.

OWEN H. WANGENSTEEN, B.A., M.D., Ph.D., Professor and Chairman, Department of Surgery, University of Minnesota Medical School.

JAMES M. WINFIELD, A.B., M.D., Professor of Surgery, New York Medical College; Chief of Staff and Attending Surgeon, Flower and Fifth Avenue Hospitals; Director of Surgery and Visiting Surgeon, Metropolitan Hospital.

ASHER WINKELSTEIN, B.S., M.D., Assistant Professor of Clinical Medicine, Columbia University, College of Physicians and Surgeons, Associate Physician in Gastroenterology and Physician in Charge, Gastrointestinal Clinic, The Mt. Sinai Hospital.

STEWART WOLF, A.B., M.D., Assistant Professor of Medicine, Cornell University Medical College; Associate Attending Physician, New York Hospital; Clinical Associate Neuropsychiatrist, Bellevue Hospital.

FIRST SESSION
THURSDAY MORNING, 12 OCTOBER 1950

9:00 A.M.

Address of Welcome—

C. J. TIDMARSH, M.D., President, National Gastroenterological Association.

9:15 A.M.

1. **"Nutritional Diseases of the Mouth and Their Differential Diagnosis".**

DR. HARRY D. FEIN

9:45 A.M.

2. **"Lesions of the Mouth".**

DR. JOHN W. RICHTER

10:15 A.M. Recess to visit the Commercial and Technical Exhibits.

10:30 A.M.

3. **"Diseases of the Esophagus".**

DR. RUDOLPH NISSEN

VISIT THE EXHIBITS

11:00 A.M.
4. "Lesions of the Esophagus".
Dr. John L. Pool

11:30 A.M.
5. "Fixed Point as a Diagnostic Aid in Lesions of Esophageal Gastric Junction".
Dr. Armistead C. Crump

SECOND SESSION

THURSDAY AFTERNOON, 12 OCTOBER 1950

1:00 P.M.
6. "Liver Injury and Liver Repair".
Drs. I. S. Ravdin and H. M. Vars

1:30 P.M.
7. "Liver Biopsy".
Dr. Emanuel M. Rappaport

2:00 P.M.
8. "Surgical Therapy for Gastrointestinal Hemorrhage in Portal Hypertension".
Dr. Louis M. Rousselot

2:30 P.M. Recess to visit the Commercial and Technical Exhibits.

2:45 P.M.
9. "Cholecystic Diseases".
Dr. Martin E. Rehfuss

3:45 P.M.
10. "The Surgical Treatment of Acute Cholecystitis".
Dr. Frank Glenn

4:15 P.M.
11. "Endocrine Stimuli of the Gastrointestinal Functions".
Dr. Franklin Hollander

THIRD SESSION

FRIDAY MORNING, 13 OCTOBER 1950

9:00 A.M.
12. "Surgical Treatment of Cancer of the Pancreas".
Dr. Alexander Brunschwig

9:30 A.M.
13. "Fibrosis of the Pancreas".
Dr. Sidney Farber

10:00 A.M. Recess.

10:15 A.M.
14. "Recent Advances in the Diagnosis and Treatment of Pancreatic Disease".
Dr. Thomas H. McGavack

VISIT THE EXHIBITS

10:45 A.M.
15. "Carcinoma of the Pancreas, Diagnosis and Treatment'.
Dr. William Barclay Parsons

11:15 A.M.
16. "Psychosomatic Aspects of Gastrointestinal Diseases".
Dr. Stewart Wolf

FOURTH SESSION

FRIDAY AFTERNOON, 13 OCTOBER 1950

1:00 P.M.
17. "Management of Duodenal Ulcer".
Dr. Ralph Colp

1:30 P.M.
18. "Ulcerative Colitis".
Dr. Burrill B. Crohn

2:00 P.M.
19. "Indications and Contraindications for Gastroscopy".
Dr. Harry Barowsky

2:30 P.M.
20. "Physiopathology of Jaundice".
Dr. Harry Shay

3:00 P.M. Recess.

3:15 P.M.

21. "Surgery and Malignancy of the Alimentary Tract".
Dr. Owen H. Wangensteen

4:15 P.M.
22. "Malignancies of the Lower Bowel".
Dr. James M. Winfield

6:30 P.M. Reception and Banquet — Hotel Statler.

FIFTH SESSION

SATURDAY MORNING, 14 OCTOBER 1950.

9:00 A.M.
23. "Modern Concepts in the Surgical Management of Diseases of the Colon".
Dr. Henry W. Cave

9:30 A.M.

24. "Psychophysiological Studies of a Female With a Large Gastric Fistula".
Dr. Asher Winkelstein

VISIT THE EXHIBITS

10:00 A.M.

25. "Evaluation of the End Results in Surgical Management of Chronic Duodenal Ulcers for the Past Twenty Years".
Dr. J. William Hinton

10:30 A.M. Recess

10:45 A.M.

26. "Allergic Manifestations in the Gastrointestinal Tract".
Dr. Albert F. R. Andresen

11:15 A.M.

27. "Newer Trends and Technics in Gastroscopy".
Dr. Leonidas H. Berry

SIXTH SESSION

SATURDAY AFTERNOON, 14 OCTOBER 1950

1:00 P.M.

28. "The Small Intestine — Clinical and Roentgenological Considerations".
Dr. Jacob Buckstein

1:30 P.M.

29. "Sources of Error in the Radiological Diagnosis of Lesions of the Gastrointestinal Tract".
Dr. J. Stauffer Lehman

2:30 P.M.

30. "Roentgenological Studies of the Mucosa of the Colon".
Dr. Franz J. Lust

3:00 P.M. Recess

3:15 P.M.

31. "Amebiasis".
Dr. I. Snapper

3:45 P.M.

32. "Carcinoma of the Stomach".
Dr. George T. Pack

SCIENTIFIC EXHIBITS

The Scientific Exhibits will be in the Exhibit Hall and will be open at the following times:

MONDAY, 9 October 1950—9:00 A.M. to 4:00 P.M.

TUESDAY, 10 October 1950—9:00 A.M. to 5:30 P.M.

WEDNESDAY, 11 October 1950—9:00 A.M. to 5:30 P.M.; 8:00 P.M. to 11:00 P.M.

THURSDAY, 12 October 1950—9:00 A.M. to 3:00 P.M.

VISIT THE EXHIBITS

BOOTH A **"Comparative Value of Gastroscopy and X-ray in 2,500 Gastroscopic Examinations".**

Drs. Leonidas H. Berry, Quentin Young, Albert L. Spaulding, and Charles L. Williams, Cook County Graduate Medical School, Provident and Cook County Hospitals, Chicago, Ill.

BOOTH B **"Carcinoma of the Esophagus"**

Drs. Earl J. Halligan, Louis L. Perkel and J. Kenneth Catlaw, Jersey City Medical Center, Margaret Hague Maternity Hospital, Jersey City, N. J.

BOOTH C **"Diaphragmatic Hernia".**

Dr. Adolph Abraham, New York, N. Y.

TECHNICAL EXHIBITORS

(Those attending the Convention sessions are urged to take advantage of the time in between the presentation of papers and sessions, to visit the technical exhibits and become acquainted with many new products and new equipment on display)

AMERICAN CYSTOSCOPE MAKERS, INC., New York, N. Y. (Booth 3), extends a cordial invitation to members and guests to visit their booth. Their new operating gastroscope and examining gastroscope of small diameter as well as other instruments of interest to the gastroenterologist will be on display.

AMES COMPANY, INC., Elkhart, Indiana (Booth 23), will be demonstrating *Clinitest, Acetest, Bumintest,* and *Hematest,* simplified tests for the detection of urine-sugar, acetone bodies, albumin, and occult blood. They will be discussing *Decholin* and *Decholin Sodium,* the standard hydrocholeretic agents for the treatment of biliary tract diseases.

J. BEEBER CO., INC., New York, N. Y. (Booths 4 and 5), is pleased to present the newest equipment used in diagnostic work, as well as the treatment of diseases: The *Raytheon Microwave Diathermy,* which is considered one of the foremost advancements in medicine; the *Brocinet-Mass Clinical Analyzer,* photoelectric colorimeter, precalibrated for the most accepted procedures; the *Mikrark Illuminator;* the *Beck-Lee direct developing Electrocardiograph; Mattern's latest X-ray Unit,* especially designed for the gastroenterologist, and many other scientific aids for the medical profession.

BILHUBER-KNOLL CORP., Orange, N. J. (Booth 28). Visit their exhibit for the latest developments on their antispasmodic-sedative, *Faloctin.* It and their medicinal chemicals, such as *Bromural, Dilaudid, Octin, Tannalbin,* etc., fill important places of useful medication in our selected field of medicine. Your visits will always be welcome.

BRISTOL-MYERS COMPANY, New York, N. Y. (Booth 14), cordially invites you to visit its booth where *Bristol-Myers'* representatives will be in attendance to extend a hearty welcome to all visitors and to answer any questions pertaining to *Bufferin, Sal Hepatica* and other famous products on display.

CAMEL CIGARETTES, New York, N. Y. (Booth 2), will feature color slides of background data from their newest research. After weekly examinations of the throats of hundreds of men and women smoking *Camel* Cigarettes exclusively for thirty days, throat specialists reported "Not one single case of throat irritation due to smoking *Camels.*"

CAMERON SURGICAL SPECIALTY COMPANY, New York-Chicago (Booth 25). Featured this year at the Cameron booth you will find the new *Omniangle gastroscope* with the coated lenses (approximately 30% more illumination) which give the greatest amount of clarity ever obtained in any flexible gastroscope. Also see the *Boris flexible esophagoscope*—the new double lighted sigmoidoscope with 4X magnification.

CIBA PHARMACEUTICAL PRODUCTS, INC., Summit, N. J. (Booth 29), invites you to visit its exhibit which will feature *Carmethose,* the organic and nonconstipating antacid for the treatment of gastric hyperacidity and peptic ulcers. Representatives in attendance will gladly answer questions on this or other Ciba products.

THE COCA-COLA COMPANY, Atlanta, Georgia (Booths 7 and 8). "Ice-cold Coca-Cola served through the cooperation and courtesy of The Coca-Cola Bottling Company of New York, Inc. and The Coca-Cola Company."

COCHRANE PHYSICIANS' SUPPLIES, INC., New York, N. Y. (Booth 36), will show latest models of instruments both foreign and domestic, including varied specialties. Featured will be the esophageal dilator set as conceived by Dr. Armistead C. Crump.

CORECO RESEARCH CORPORATION, New York, N. Y. (Booth 9). The *Coreco* Camera is designed to photograph all surface areas of the body, from 1 to 1 close-up pictures to half-body size—and all cavities of the human body, such as mouth, throat, ear, nose, vagina, and rectum. The Camera carries its own specially developed, fully color-corrected bulb and a mechanism for complete control of its color temperature and exposure within the camera itself. There is an automatic view finder synchronized with the automatic camera mechanism to permit viewing until a fraction of a second before exposure. The Camera provides for automatic focusing.

EDER INSTRUMENT CO., Chicago, Ill. (Booth 38), will show an extensive line of Gastroscopic equipment. Several of their items will be shown for the first time, and they will be of interest to all the members and guests at their booth.

THE HARROWER LABORATORY, INC., Jersey City, N. J. (Booth 30). Their exhibit has three main points of interest: 1. Gastroscopic pictures illustrating the coating action of *Mucotin*, the "Council Accepted" mucin antacid. 2. Schematic presentation of the physiological action of *Prulose*, a corrective laxative. 3. The role of the Kreb Cycle and *Hematocrin* in energy metabolism is graphically shown. Reprints and samples will be available at this booth.

IVES-CAMERON COMPANY, INC., New York, N. Y. (Booth 33), have several new drugs of especial interest to the gastroenterologist. *Monitan, I.V.C.*, for the use of treating conditions of impaired fat emulsification and absorption as well as *Oxsorbil, I.V.C.*, a superior choleretic-cholagogue and fat emulsifier are among those to be featured. Trained representatives will be able to discuss the indications and pharmacological action of their new therapies much discussed in the literature today.

KEGAN LABORATORY, INC., Englewood, New Jersey (Booth 11). *Phytotone* is a palatable, stable, high potency source of *natural* Vitamin C together with Rutin, Pectin, and other nutritional substances derived from selected herbs and fruits. In peptic ulcer therapy physicians will find its high content of *natural* Vitamin C (120 mg. per fl. oz.) and Rutin particularly beneficial. Taste-test it at Booth 11.

LANE LIMITED, New York, N. Y. (Booth 12), will feature the merits and quality of *Lords* cigarettes and *Lords* pipe mixture. Both products are guaranteed to contain less than 1% nicotine. Backed by over three generations of tobacco manufacturing, these products will demonstrate the results of an exclusive process allowing extraction of nicotine and tars without medicants and chemicals, yet not sacrificing taste, body, or flavors of the tobacco.

J. B. LIPPINCOTT COMPANY, Philadelphia, Pa. (Booth 24), presents an interesting and active exhibit of professional publishing. With the "pulse of practice" centering in an advisory editorial board of active clinicians who constantly review the field, current and coming trends in medicine and surgery are known continually. On the studied recommendations of these medical leaders, Lippincott Selected Professional Books are undertaken.

MALLON, Division of DOHO CHEMICAL CORPORATION, New York, N. Y. (Booth 1), is introducing their new liquid topical anesthesia, *Rectalgan*, for relief of pain and itching in hemorrhoids and pruritus; also following perineal suturing in obstetrical and gynecological work. This new therapy enjoys many advantages over the outmoded suppositories and ointments. Their representatives will be happy to explain, in detail, the workings of this medication.

THE NATIONAL DRUG COMPANY, Philadelphia, Pa. (Booth 35), cordially invites you to visit their booth where *Resion* and *Resinat* will be the featured preparations. *Resion*, an intestinal adsorbent combining the properties of ion exchange and physical adsorption and *Resinat* (Council Accepted), newly accepted therapy for peptic ulcer will be on display and representatives will be in attendance to discuss with physicians the various aspects of these two outstanding products.

NORGINE LABORATORIES, INC., New York, N. Y. (Booth 13), will exhibit three pharmaceutical products of particular interest to the gastroenterologist, i.e.: *Enzypan*—a multivalent digestive enzyme compound; *Spasmacol*—a bulk laxative combined with a spasmolytic for spastic constipation, and *Gamatran*—a synthetic spasmolytic for sustained-action spasmolysis. Samples, literature and information will be supplied by Norgine representatives.

PICKER X-RAY CORPORATION, New York, N. Y. (Booth 39), invites you to visit an exhibit of the latest developments in high-powered diagnostic x-ray equipment. A staff of engineers and technicians will be in attendance to demonstrate the apparatus and furnish all pertinent data.

A. H. ROBINS CO., INC., Richmond, Va. (Booth 10), is featuring *Entozyme*, triple-enzyme digestant with the unique "peptomatic" tablet; the sedative-antispasmodic, *Donnatal;* the anti-rheumatic, *Pabalate;* and *Robitussin*, Robins' rational formulation for cough therapy. Robins' Professional Service Representatives will welcome the privilege of discussing with physicians attending, these and other products in the company's line of prescription specialties.

WILLIAM H. RORER, INC., Philadelphia, Pa. (Booth 17). *Maalox-Rorer*, a colloidal suspension of Magnesium and Aluminum Hydroxides is a new antacid preparation, indicated for routine use in treatment of peptic ulcer. *It eliminates the constipation difficulties* which so often accompany administration of aluminum hydroxide gel, yet retains the fine antacid properties of that drug. The taste of *Maalox-Rorer* is particularly well received by patients.

RYSTAN COMPANY, INC., Mount Vernon, N. Y. (Booth 22), will introduce the new chlorophyll preparation for the medical management of peptic ulcer—*Chloresium Powder*—which was reported in the May 1950 issue of THE REVIEW OF GASTROENTEROLOGY. This preparation combines the tissue-stimulating properties of water-soluble chlorophyll with a highly persistent coating material, dehydrated powdered okra, plus the proven antacids, aluminum hydroxide and magnesium trisilicate.

SANDOZ CHEMICAL WORKS, INC., New York, N. Y. (Booth 26), will feature the following: *Cafergone*—the first effective oral preparation for the treatment of migraine and related headaches. Other products displayed are *Bellergal, Belladenal, Mesantoin, Dihydroergotamine (D.H.E. 45)* and *Ipesandrine*. A new handbook listing our products will be available and representatives in attendance will gladly answer any questions about these and other Sandoz products.

SARATOGA SPRINGS AUTHORITY, Saratoga Springs, N. Y. (Booth 19). This exhibit consists of a new knotty pine display emphasizing treatments with use of naturally carbonated mineral water at the New York State-owned Saratoga Spa at Saratoga Springs, N. Y. Recreational features are also described. Literature will be available.

SCHENLEY LABORATORIES, INC., Lawrenceburg, Ind. (Booth 32). We believe Dr. Sippy would approve of *Titralac*, a new and unique antacid preparation highlighted at the Schenley Laboratories exhibit. Combining the buffering action of glycine with the neutralizing properties of calcium carbonate, one *Titralac* tablet provides the acid-inactivating power of an 8 ounce glass of milk. Conveniently administered, exceptionally palatable, *Titralac* is useful in hyperacidity and peptic ulcer management, especially attractive to allergic patients.

G. D. SEARLE & CO., Chicago, Ill. (Booth 31), cordially invites you to visit their booth where their representatives will be happy to answer any questions regarding Searle Products of Research. Featured will be *Dramamine* for the prevention and active treatment of motion sickness; *Alidase*, for hypodermoclysis; *Ruphyllin*, for abnormal capillary fragility; *Hydryllin*, new and effective antihistaminic, as well as such time-proven products as Searle *Aminophyllin* in all dosage forms, *Metamucil, Ketochol, Floraquin, Kiophyllin, Diodoquin, Pavatrine*, and *Pavatrine with Phenobarbital*.

SENTRAL LABORATORIES, INC., Cedar Rapids, Iowa (Booth 15), will exhibit the following three products: A protein digest in liquid form, also called an amino acid product; a wheat germ oil concentrate; a tablet containing the major enzymes of digestion (called a *Panenzyme*).

E. R. SQUIBB AND SONS, New York, N. Y. (Booth 21).

FREDERICK TROUT COMPANY, Mount Vernon, N. Y. (Booth 6), will feature *Combichole*, a combination of two chemically pure bile acids for Hydrocholeresis and fat Emulsification, also *Calsamate*, an Analgesic buffered with Calcium Gultamate to prevent pyrosis and Ascorbic Acid as replacement therapy.

U. S. VITAMIN CORPORATION, New York, N. Y. (Booth 37). New lipotropic therapy . . . *Methischol* . . . original combination of four proven lipotropic agents: choline, methionine, inositol and liver extract. Also, taste for yourself the new and different sodium-free salt substitute, which tastes like salt, looks like salt and sprinkles like salt. Professional samples on their full line of nutritional specialties will be distributed.

WINTHROP-STEARNS, INC., New York, N. Y. (Booth 20), extends a cordial invitation to visit its booth, where representatives will be on hand to serve you. Featured products will be *Creamalin* ® non-alkaline, non-absorbable antacid; *Tricreamalate* ® Creamalin (aluminum hydroxide) plus magnesium trisilicate; *Pluraxin* ® high potency, therapeutic formula multiple vitamin capsules; *Pluravit* ® drops, dispersible 8 vitamin liquid, including *Drisdol*, pleasing citrus flavor, stable, easily administered.

WYETH INCORPORATED, Philadelphia, Pa. (Booth 27). *Hydase*—Wyeth's highly purified hyaluronidase and council-accepted *Amphojel*—for the symptomatic relief of peptic ulcer will be featured along with such widely prescribed ethical specialties as *Kaomagma* with Pectin, *S.M.A., Wychol, Pondets, Beplete, Purodigin and Propion Gel*. Trained representatives will be on hand to supply literature and samples of outstanding therapeutic agents.

F. E. YOUNG & CO., Chicago, Ill. (Booth 34). Young's Dilators are used in the treatment and prevention of contracted anus, particularly following hemorrhoidectomy, as an aid in perineal dissection and in the repair following delivery. Physician's Pharmacal Service is a comprehensive listing of ethical products in table form for easy reference, kept up to date by monthly supplements and being loose leaf, new pages easily replace former pages. *Sulf-A-Test, Young's PSP Test Set* and *Young's Albumin Test* will be demonstrated.

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You want.....FAST HEALING

Creamalin relieves ulcer pain in minutes because it is truly *amorphous* aluminum hydroxide, the form emphasized by Sollmann[1] as being most soluble. Greater solubility makes Creamalin rapidly available for neutralization. Also, Creamalin is a *reactive* gel. Reactive gels exhibit greater acid-combining power.[2]

Creamalin heals the lesion in 7 to 10 days because it is *acid-soluble* aluminum hydroxide, the nonabsorbable antacid which provides sustained neutralization at pH 4 to pH 5. Healing is fast because Creamalin inhibits pepsin activity and is "mucosal adherent." Tablets and liquid.

1. Sollmann, T.: A Manual of Pharmacology. Philadelphia, W. B. Saunders Co., 7th ed., 1948, p. 938.
2. Batterman, R. C., and Ehrenfeld, L.: Gastroenterology, 9:141, Aug., 1947.

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Victor T. McGrattan, New York, N. Y., Fellow; William E. Bippus, West Palm Beach, Fla., Associate Fellow; A. Strelinger, Elizabeth, N. J., Associate Fellow; Benjamin J. Macchia, Jersey City, N. J., Associate Fellow; Leon Bader, New York, N. Y., Associate Fellow; George B. J. Glass, New York, N. Y., Associate Fellow.

The matter of the Constitution and By-Laws was taken up where the Board had left off at the previous meeting and the draft submitted by Dr. Tidmarsh was accepted. The entire Constitution was to be put into proper order and form and submitted to the membership at large for adoption.

At the meeting of the Executive Board held in New York City on 21 May 1950, the Association was advised of the death of Dr. Samuel D. Weaver of Dallas, Texas. The Secretary was instructed to send the usual letter of condolence.

The following new members were elected to the National Gastroenterological Association: Drs. George Francis Miller, Boston, Mass., Member; Isadore Irving Cash, Milwaukee, Wisc., Member; Thomas C. Wilkinson, Pittsburgh, Pa., Member; Michael Q. Hancock, Belmar, N. J., Member; Herbert I. Puller, Roselle, N. J., Member; Edward F. Sciorsci, Hoboken, N. J., Member; Herbert Silberner, Newark, N. J., Member; Irvin Sussman, Bridgeton, N. J., Member; Solomon E. Gittleman, Brooklyn, N. Y., Member; Barnet I. Romm, Brooklyn, N. Y., Member; Harry O. Maryan, Chicago, Ill., Member; Edward J. Krol, Chicago, Ill., Member and Brian T. Shorney, St. Croix, Virgin Islands, Member.

Upon presentation of the necessary qualifications the following were advanced as indicated: Ludmilla F. Brown, New York, N. Y., Associate Fellow; Alfred J. Cantor, Flushing, N. Y., Associate Fellow; A. I. Friedman, Hackensack, N. J., Associate Fellow; Walter Freymann, West New York, N. J., Associate Fellow; Samuel M. Gilbert, Newark, N. J., Associate Fellow; Paul H. Moore, Hollywood, Calif., Associate Fellow; P. M. Moriarty, Chicopee, Mass., Associate Fellow; Helmuth Nathan, New York, N. Y., Associate Fellow; H. F. Pohlman, Middletown, N. Y., Associate Fellow; L. Pulsifer, Rochester, N. Y., Associate Fellow; Louis Rosenblum, New York, N. Y., Associate Fellow; Jerome Weiss, New York, N. Y., Associate Fellow.

The matter of the length of time necessary for printing papers submitted for publication to THE REVIEW OF GASTROENTEROLOGY was discussed and it was pointed out that at present papers presented before the Convention and the Course take precedence over all other papers submitted, to be followed by papers submitted by members of the Association and by non-members.

With this in mind it was suggested the size of the Journal be increased and also that papers be limited in size in order to accommodate more papers per issue. The matter was referred to the Editorial Committee for their consideration.

At the last meeting of the Executive Board of the National Gastroenterological Association held on 20 June 1950, in New York City, routine reports were received.

Upon recommendation of the New Jersey Chapter the following applications were accepted and ratified as indicated: Dr. Paul J. Miranti, Jersey City, N. J., Member and Dr. Leo P. Schultz, Allendale, N. J., Member.

Dr. LeRoy D. Miller, Brooklyn, N. Y., was elected a Member at large of the National Gastroenterological Association.

Upon submitting the necessary qualifications, Dr. Helmuth Nathan of New York City was advanced to Fellowship.

THIRD SURGICAL COURSE ON PATHOLOGY OF THE DIGESTIVE TRACT

The Third Surgical Course on Pathology of the Digestive Tract will be held in Madrid, Spain in November 1950.

Registration for the Course will take place between the 15th of September and the 15th of October 1950, and further information may be obtained by writing the Secretary, Dr. C. Gonzalez Bueno, Calle de Villanueva, Num. 10, Madrid, Spain.

INTERNATIONAL ACADEMY OF PROCTOLOGY

At the Second Annual Meeting of the International Academy of Proctology held in San Francisco on 23 June 1950, the following officers were elected for the year 1950.

Dr. Caesar Portes, Chicago, Ill., President; Dr. H. A. Springer, Cincinnati, Ohio, President-elect; Dr. Edgar M. Scott, Jr., Birmingham, Ala., First Vice-President; Dr. Norman I. Albert, Johnston City, Ill., Second Vice-President; Dr. Donald C. Collins, Hollywood, Calif., Third Vice-President; Dr. Alfred J. Cantor, Flushing, N. Y., Secretary; Dr. William Lieberman, Brooklyn, N. Y., Treasurer.

Dr. Francis S. Adams, Pueblo, Colorado; Dr. Carroll J. Bellis, Long Beach, Calif.; Dr. Harry A. Gussin, Chicago, Ill.; Dr. Raymond S. Johnston, Kearny, Nebraska; Dr. Edward Krol, Chicago, Ill.; Dr. Paul Lahvis, Gowanda, N. Y.; Dr. William W. Meissner, Buffalo, N. Y. and Dr. Evan C. Reese, Stroudsburg, Pa. are the members of the Executive Board.

The next Annual Meeting of the International Academy of Proctology will be in Atlantic City, N. J., during the meeting of the American Medical Association.

BOOK REVIEWS

LIPIDOSES—DISEASES OF THE CELLULAR LIPID METABOLISM. Siegfried J. Thannhauser, M.D., Ph.D., Associate Professor of Medicine, Tufts College Medical School; Associate Physician-in-chief, Joseph H. Pratt Diagnostic Hospital, Boston, Mass. Edited by Henry A. Christian, A.M., M.D., LL.D., Sc.D. (Hon.), M.A.C.P., Hon. F.R.C.P. (Can.), D.S.M. (AMA). Boston, Mass. Second edition. 605 pages (including 10 page index). Oxford University Press, New York, 1950 Price $12.00.

This excellent encyclopedic work is reprinted from Oxford Loose-Leaf Medicine with the same page numbers as in that work. The first edition of this reference work appeared in 1940 while basically, not many changes have taken place in the conception of the pathogeneses of the several "lipidoses" extensive revision is evident in the sections on xanthomatous biliary cirrhosis and familial hypercholesteremic xanthomatosis, idiopathic hyperlipemia and eosinophilic xanthomatous granuloma including Schüller-Christian syndrome and essential xanthomatosis of the normocholesteremic type. *Adult* Niemann-Pick's Disease; "Gargoylism"-Hurler-Pfaundler syndrome; Gaucher's Disease; reticular and histiocytic sphingomyelinosis (lipid cell splenohepatomegaly or Niemann-Pick's phosphated lipoidosis) infantile form; are expertly discussed in great detail.

Brief discussions are included on lipoid proteinoses, Urbach and Wiethe (1929), and Oppenheim's (1929) necrobiosis lipoidica diabeticorum which Urbach (1932) also described.

Discussion of both the infantile and adult types of Gaucher's reticular and histiocytic cerebrosidosis are presented in Part IV, in 74 pages (pp. 456-530).

Glycogen storage disease (Von Gierke) or "hepato-nephromegalia glycogenica" (1929) instances of which had been previously reported by Wagner and Parnas (1921) and by Snapper and Van Creweld (1928) (pp. 314-329), is adequately and satisfactorily presented. The bibliography with an extensive list of references which follows each section should be most helpful to the investigator and student of the subject.

This exhaustively complete reference work on the "Lipidoses" is the best book available on the subject, and is most enthusiastically recommended as the most authoritative and informative work in print, available to all who wish to be well-informed.

OPERATIONS OF GENERAL SURGERY. Thomas G. Orr, M.D., Professor of Surgery, University of Kansas School of Medicine, Kansas City, Kansas. Second Edition. 890 pages with 1700 step-by-step illustrations on 721 figures. W. B. Saunders Company, Philadelphia, Pa., 1949. Price $13.50.

This second edition of Dr. Orr's volume on surgery surpasses the original publication. The publishers spared no expense in making this volume a real teaching text. The third year medical student who is being initiated into the mysteries of clinical minor surgery will not have any excuse that he is unable to grasp the principles of minor surgery. Later as he becomes a senior, clinical assistant, interne and resident, he will find the step-by-step illustrations and explanations in minor surgery, major surgery and the specialties clear and to the point.

It is highly recommended as a practical guide to the student as well as the surgeon. The latter will find that the illustrations and text will aid him in his chosen work.

CLINICAL NEUROLOGY. Bernard J. Alpers, M.D., Professor of Neurology, Jefferson Medical College, Philadelphia; Neurologist to the Jefferson, Pennsylvania and Wills Hospitals, Philadelphia. Second Edition. 846 pages with 240 illustrations. F. A. Davis Co., Philadelphia, Pa., 1949. Price $9.50.

This excellent work on clinical neurology by an experienced teacher and neurologist of note, has been extensively revised including poliomyelitis, epilepsy, meningitis, syphilis of the nervous system, peripheral nerve injuries and cranial trauma.

Considerable space is given to the examination of the nervous system; the interpretation of neurological symptoms and signs and topical diagnosis of nervous disease. Adequate informative discussion of headache, vertigo, coma and pain appears in Chapter 4. Careful consideration is given to neuritis and the neuralgias including Fothergill's Disease, Dejerine Sotta's hypertrophic interstitial neuritis, pellagra neuritis and alcoholic neuritis. Diseases of the spinal cord are quite thoroughly discussed, including hypertrophic spinal pachymeningitis, extradural abscess; arachnoiditis; herpes zoster or posterior poliomyelitis—primary viral radiculitis and ganglionitis and secondary symptomatic zoster due to irritation of roots associated with many disorders. The primary form of zoster or posterior poliomyelitis is due to a filterable virus which has been isolated. The condition is a myelitis involving the posterior root and its ganglion and the posterior horn of the cord. The posterior root and its ganglion are infiltrated by lymphocytes and sometimes plasma cells. Hemorrhage and necrosis may sometimes be found in the ganglion. Ophthalmic herpes is due to involvement of the Gasserian ganglion and its root. In 5.7 per cent of the cases, an encephalitis develops with herpes ophthalmicus. This is one of the best available discussions on the subject in recent literature.

Anterior poliomyelitis, spinal fractures, syringomyelia, Charcot-Marie-Tooth atrophy, subacute combined degeneration and Marie-Strümpell Disease are briefly, but instructively presented.

The author gives careful consideration to "Disease of the Meninges", "Encephalitis, syphilis of the brain, and vascular disease of the brain.

Tumors of the brain, abscess of the brain, cranial trauma, epilepsy and convulsive states, extrapyramidal diseases, multiple sclerosis, nuclear amyotrophies and myopathies including myasthenia gravis, familial periodic paralysis, and amyotrophic lateral sclerosis are expertly discussed. The closing chapters include the degenerative diseases, amaurotic family idiocy, Schilder's disease, Little's disease, porencephaly, and Marchiafava's Disease; congenital and developmental disorders and diseases of the skull and vertebrae.

In this reviewer's opinion this textbook is one of the best available volumes on clinical neurology for the medical student, the general practitioner, and the clinician. It is enthusiastically recommended as a desk volume for ready use.

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WHEN YOU'RE DAYDREAMING perhaps you, like most people, find yourself doodling pictures of the things you want most.

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Although these patients have no apparent organic basis for their complaints, they are ill and merit attention.

In functional disorders, response to stress is effected via both branches of the autonomic nervous system. Therefore, treatment consists, where possible, in removal of the emotogenic factor (practical psychotherapy) and the "partial blockade" of the efferent autonomic pathways. The family physician is well-qualified to help these patients; his advice will do much to achieve the desired change in habits and to avoid unhealthy situations.

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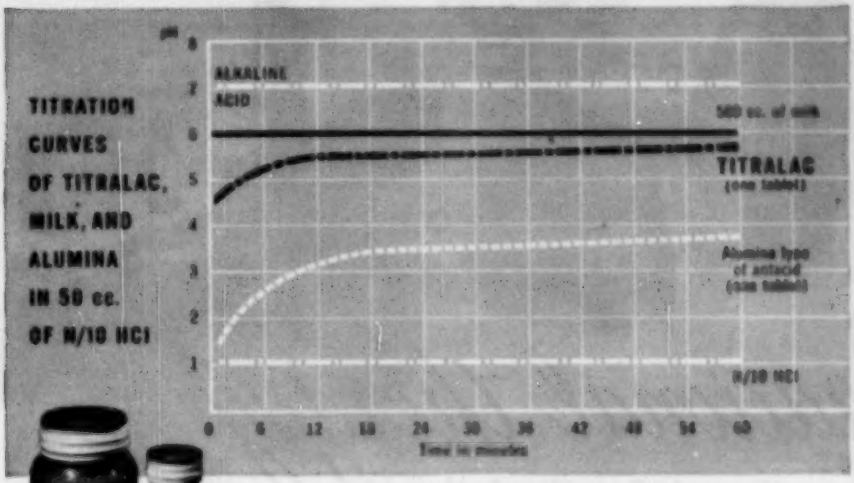

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Gastroenterologists have long endorsed the use of milk, when practicable, for its ideal acid-converting power and buffering capacity.[1, 2] In a recent comprehensive paper, Aaron[3] and others[4, 5, 6] express a preference for calcium carbonate as the antacid to be employed.

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TITRALAC tablets are supplied in bottles of 100 and convenient-to-carry packages of 40. TITRALAC powder is also available, in 4-oz. jars.

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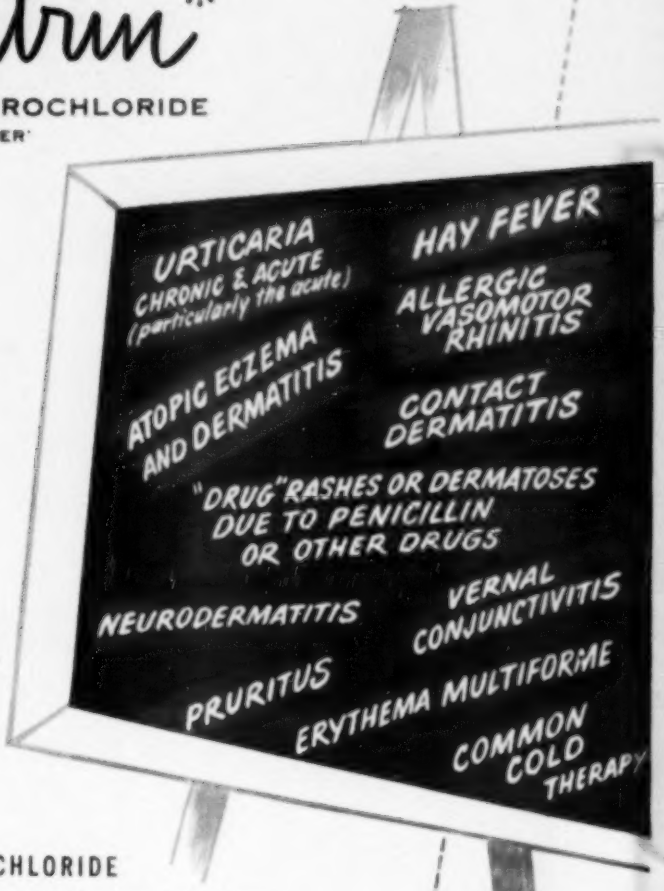

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REFERENCES

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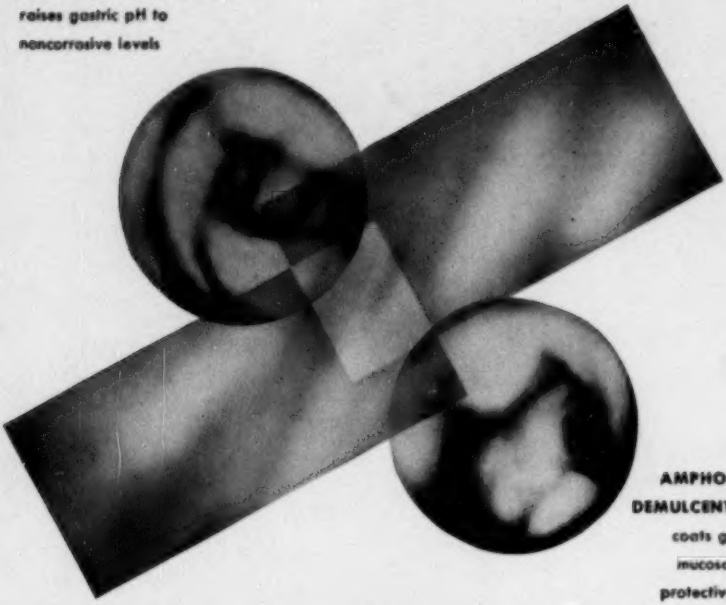

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